BRAIN.

APRIL, 1881.

Original Articles.

THE LATE LORD CHIEF JUSTICE OF ENGLAND ON LUNACY.

BY JOHN CHARLES BUCKNILL, M.D., F.R.S.

One has recently passed from among us in the full ripeness of years and of honours, who has left a deep impression upon our memories, of the force and versatility of his character, and of the decided and sometimes original opinions which he had formed and maintained upon many of the subjects which fell within the scope of his wide influence. For in many of the most interesting dramas which for many years past have been presented on the political or the forensic stage Sir Alexander Cockburn played one of the leading parts, and it especially fell to his lot to be engaged in contentions whose issues, if they have not greatly promoted the science of psychology, have at least gone far to shape and determine the practical question of the relations of the insane with society and its laws, both with regard to their exemptions and their disabilities.

Among the former, the most remarkable and memorable was his defence of McNaughten for shooting Mr. Drummond, on the plea that he was not guilty on the ground of insanity. This *cause célèbre* has been worn threadbare by discussion, but in looking over Cockburn's very able advocacy we cannot fail to notice his generous recognition of the great value of medical knowledge and experience in determining such difficult questions as the one before the Court; a fixed state of feeling and belief which, to the last days of his long life, he rarely

failed to express whenever the opportunity offered, in observations which form a notable contrast to such remarks disparaging to medical science as are even now sometimes heard in Courts; remarks which tend to show, if they mean anything, that great lawyers sometimes have not the wit, or will not take the pains, to distinguish real science from false pretensions to it. In McNaughten's defence Mr. Cockburn said: "It was now placed beyond doubt that madness was a disease of the body, the result of morbid organization, and that its nature was to be precisely and accurately ascertained by those only who had made this disease and its pathology the object of long reflection and diligent investigation. The discoveries of modern science had thrown much light upon this subject, and many of the positions laid down by Lord Hale and the other authorities of former times were left liable to very great objection and doubt."[1]

In addition to the stress which he laid upon the value of scientific knowledge, a remarkable feature in his defence of McNaughten is apparent in his early recognition of the element of loss of self-control. He undertook to prove to the jury that the prisoner "was the creature of delusion and uncontrollable impulse, which took away from him the character of a responsible being." This term "uncontrollable impulse" has since been very greatly misused and abused, inasmuch as it has been attributed to offenders who had no other characteristics of insanity; and, as we shall see, Cockburn subsequently adopted the better term of "absence of the power of self-control." While we must repudiate the overstrained doctrine that uncontrollable impulse is in itself and by itself, a form of insanity, we must recognise the fact that the quality is essential to the commission of an offence by a madman. Whether we say that the man is impelled to do a thing by his madness, or whether we say he did that which his mad thoughts led him to do in "the absence of self-control," or as another great judge has put it, that he did that in his madness which he could not help doing, we have the same thought with some little variety of expression. If we could know that a person having a delusion could so have controlled himself as not to

[1] The 'Times,' March 6th, 1843.

have indulged or acted upon it, our view of his responsibility would be very different to that which we must take in regard to "a creature of delusion" deprived of all self-control. The practical difficulty, of course, is that of deciding when, in consequence of disease of the organism, a man is deprived of self-control or has an uncontrollable impulse, a difficulty certainly not less than that of deciding when a person of unsound mind does or does not retain the knowledge of right and wrong with regard to his actions; but why the difficulty should be thought by our legal authorities to be insurmountable in the one case but not in the other, it is not easy to understand. We must briefly refer to Cockburn's opinion on this important point, and also to that of his compeers who were not able to concur in it.

Thirty-one years after the McNaughten trial, the counsel for the defence, now become Lord Chief Justice of England, repeated the opinions maintained in that trial, in all the maturity of his judgment and with all the weight of his authority, in his Memorandum to the Chairman of the Select Committee of the House of Commons on the Homicide Law Amendment Bill. In this important document he says:

"As the law, as expounded by the Judges in the House of Lords, now stands, it is only when mental disease produces incapacity to distinguish between right and wrong, that immunity from the penal consequences of crime is admitted. The present Bill introduces a new element, the absence of the power of self-control. I concur most cordially in the proposed alteration of the law, having been always strongly of opinion that, as the pathology of insanity abundantly establishes, there are forms of mental disease in which, though the patient is aware he is about to do wrong, the will becomes overpowered by the force of irresistible impulse, the power of self-control, when destroyed or suspended by mental disease, becomes (I think) an essential element of responsibility."

There would seem to be a clerical or printer's error in this last sentence, the context clearly showing that the writer intended to say that the suspension of the power of self-control was an element of irresponsibility. And no doubt it is; the difficult question remaining to be solved as to what are the forms

or degrees of mental disease in which the power of self-control is suspended or destroyed. For instance, in general mania, with or without delusion, the power of self-control is thus suspended; but is this the case also in what is called homicidal mania, as it is described without the group of symptoms which usually accompany diseases affecting the mind? It is much to be wished that the Chief Justice had expressed his opinion more fully and clearly on this most important point. In a subsequent passage, however, towards the end of his memorandum he does connect other symptoms of insanity, that is to say delusions, with the loss of self-control in such a manner that it must be inferred that in the passage already quoted, he did not intend to endorse the dangerous and illogical theory of so-called moral insanity. In this later paragraph he says:

"I have already expressed my concurrence in the proposed provisions as to the effect of insanity, except so far as the proposed legislation is partial, as limited to the case of homicide. But there is one general provision on this subject to which I must strenuously object; it is that, 'if a person is proved to have been labouring under any insane delusion at the time when he committed homicide, it shall be presumed, unless the contrary appears to be proved, that he did not possess the degree of knowledge or self-control hereinbefore specified.' The pathology of insanity shows that the mind may be subject to delusions, which do not in any degree affect the moral sense or the will as regards the power of self-control. The mere existence of mental delusion ought not to affect the decision as to the power of self-control, unless the nature of the delusion be such as would legitimately lead to the inference that the power of self-control was wanting. The question is one which should be decided by all the circumstances, independently of any presumption one way or the other."

It will be seen, when we come to comment upon this Chief's most important judgment in a civil cause, how consistent he was in his views of what we may perhaps be permitted to call the autonomy of delusion. It may be that his views were extreme in this regard both in criminal and civil law. Correct in his fact that insane delusion may exist without the loss of self-control, he scarcely seems to have given weight enough

to the importance of delusion as evidence of insanity more or less general; and we venture to assert that where a man who has committed a homicide is proved to have been labouring under any insane delusion at the time, the inference will inevitably present itself that with the loss of the power of reasoning the man has also lost the power of self-control. Whether in such cases the legal burden of proof should be shifted, as proposed by the framers of the Bill of 1874, is quite another and an important question. It is possible that an insane delusion may not affect a particular action; but seeing that an insane delusion indicates grave mental disease, and that it is impossible to say how far its influence extends and where it stops, there must always exist great doubt as to any action being free from its influence. It must at least be extremely difficult to prove that any deliberately wrong, and especially any penal action, was not done under the influence of the delusion. Therefore this shifting of the burden of proof proposed would be tantamount to making the proof of delusion exonerate the crime. One cannot get on without presumptions, and many of them, in deciding such difficult and often recondite questions as those depending upon the influence of diseased mind upon conduct; and if the presumption of the wide-extending influence of insane delusion be admitted, it would surely be more wise in conception and simple in practice to extend its exonerating influence than to change the burden of proof in the manner proposed. The recent Royal Commission of four pre-eminent lawyers employed to draw up the Criminal Code decided against the section of the Criminal Code Indictable Offences Bill of 1878 which recognised as an excuse the existence of an [insane] impulse to commit a crime, on the ground that the test proposed [namely the supposed inefficiency of the greatest and most immediate punishment] for distinguishing between such a state of [insane] mind and a criminal motive would not be practicable or safe. In the draft Criminal Code drawn up by the Royal Commissioners, Blackburn, Barry, Lush, and Stephen, all reference to loss of control is omitted, and the existing law with regard to the pertinence of delusion to the crime is left as it was; only it is proposed newly to provide that any insane

delusions, including, of course, such as are not pertinent to the crime, may be given as evidence that the offender was in such a condition of mind as to enable him to be acquitted on the ground of insanity; that is to say, that he was labouring from natural imbecility or disease affecting the mind to such an extent as to be incapable of appreciating the nature and quality of the act, or that the act was wrong. Without doubt it is extremely difficult to formulate such a legal doctrine as that of the loss of self-control as an excuse for crime, so that it shall not be dangerous to society; but seeing that the knowledge of right and wrong can have no effect on the commission or omission of crime, unless the agent has the power to control himself so as to commit or omit the action which constitutes a crime, and seeing that madness does enormously affect this power, we cannot but think that the late Chief Justice came far nearer to a right and satisfactory solution of this difficult problem in the objections and suggestions of his memorandum than the Royal Commissioners.

In the letter which in June 1879 the Chief Justice addressed to the Attorney-General on the Criminal Code Bill, he has criticised the law as it exists, and its proposed alterations, in a manner which must increase the difficulties which judges cannot fail to experience in charging juries whenever the plea of "not guilty, on the ground of insanity," is put forward. His own opinions, however, are not so clearly expressed as to leave it without doubt what he really meant, especially on the extremely important question of homicidal insanity, or of moral insanity generally. In a recent work on insanity ('The Factors of Unsound Mind,' by Dr. Guy), his authority is quoted from this letter in support of the theory of "instinctive or impulsive homicidal monomania," and his language is certainly open to the interpretation which this author puts upon it. Thus he says:—"Among the functions of the human mind liable to be perverted by disease is, as all scientific writers on insanity are agreed, the human will, which sometimes becomes the slave of maniacal impulses, which it is unable to resist. Among the different forms of madness by which the will is liable to be thus affected, is that which is known by the term of homicidal mania, or, when it impels a person to self-destruc-

tion, suicidal mania. That the will is liable to be thus maniacally affected, and so to be swayed by impulses which it is unable to resist, is a point on which writers on mental pathology are agreed." "The question whether, under the influence of mental disease, the human will may become subject to impulses which it is unable to resist, and upon which even the fear of death will not operate as a restraint, is not one for lawyers to dispose of dogmatically, as they too often do, but one which, as a question of pathological science, it is for men conversant with that science to decide." "The question whether *mania, accompanied by insane impulse,* might afford a defence, was not submitted to the Judges [in the McNaughten case], or involved in their answers."

Now it may be argued that these passages, with the context, affirm the doctrine of insanity of the will, or at least that they affirm the existence of homicidal and suicidal monomania, as dependent upon insane impulse alone. But it is to be noted that the Chief Justice invariably speaks of these affections as *maniacal;* thus, "the will becomes the slave of maniacal impulses," and "the will is liable to be maniacally affected," and "the question is whether mania accompanied by insane impulses," &c. Nowhere does he say that the will swayed by impulses unaccompanied by the symptoms of mania, is a form of insanity, or might afford a defence. But those writers who maintain the existence of homicidal and suicidal, or of the stealing, or ravishing, or house-burning forms of so-called moral insanities, do describe them as unaccompanied by the general symptoms of mania, mental and physical. Of such forms of insanity a considerable number of "writers on mental pathology" utterly deny the existence, although all such writers will, and do, undoubtedly admit that such conduct accompanies real mania. Seeing therefore that the Chief Justice asserts that his statement of facts is a "point on which writers on mental pathology are agreed," it may fairly be argued that by the impulsive affections of the will which he describes, he meant the headstrong conduct of the real maniac, which no one denies, and therefore that he was not a convert to the new doctrine of moral insanity, which never yet has withstood the trial of experience or the test of argument.

That will-o'-the-wisp, the human will, has misled metaphysicians and moralists often enough, to make us heartily desire that at least grave and reverend Judges should be blind to its glamour. And by omitting such speculations as that "the human will becomes the slave of maniacal impulses," "which it is unable to resist," &c., we may gather that the Chief Justice intended to express his conviction that a man having no delusions might be in a maniacal condition, accompanied by insane impulses to the commission of offences against the law, and that the proof of such a condition ought to be a valid defence. This "is a point on which writers on pathology are agreed," it being understood that the maniacal condition connotes a well-known group of symptoms called mania, in persons who are properly said to be "maniacally affected."

This is "the question which was not submitted to the Judges, nor involved in their answer," and upon which the late Chief Justice expressed his opinion in such metaphysical arguments, as leave his meaning needlessly obscured, but which lead to the conclusion, as entertained by him, that a maniac, even without delusion and with knowledge of right and wrong, is nevertheless irresponsible.

This great Judge has left the mark of original thought and decision still more deeply impressed upon the civil than on the criminal side of lunacy in the Courts. As an advocate, one of his most brilliant efforts was made in his successful opposition to his distinguished opponent Sir Frederick Thesiger, in the cause of Sefton *v.* Hopwood, tried before Mr. Justice Cresswell at the Lancashire Assizes in 1855. The Earl of Sefton propounded the will on behalf of the younger children of the testator, his father-in-law, and Mr. Cockburn opposed it on behalf of the elder son, who had been disinherited. Cockburn admitted that in this case there was no evidence of delusion, which in McNaughten's case he had followed Erskine in declaring to be the essential feature of lunacy. But he reasoned, and he proved that there was abundant evidence of loss of memory and of confusion of thought extending, as by his masterful eloquence he persuaded the jury to believe, to complete testamentary incapacity. In this trial, as in McNaughten's, he gave the full meed of commenda-

tion to the medical evidence, and justly so, for the medical attendant of the great county family whose dearest interests were involved had behaved with resolution under trying circumstances in refusing to witness the will, and had given his evidence with a judgment and temper which could not fail to influence the jury towards his honest and well-considered opinion.

In the following year Sir Alexander Cockburn led in maintaining the validity of the will in the great Swynfen case. But the arguments or the statements of opinion expressed by advocates have perhaps their greatest permanent value as preparations or training of the judicial mind, upon which the determination of the law eventually depends. It may be interesting and not unprofitable in such a man to trace the arguments of the barrister in the judgments of the Judge, but the more important duty of endeavouring to estimate the full nature of the more important of these judgments must check diffuseness on such matter as does not bear the stamp of authority.

In Banks *v.* Goodfellow, the matured opinions of the Chief Justice are expressed on insanity in its relation to testamentary capacity, and this judgment is remarkable on account of its forming one of the rare instances in which all accepted precedents of legal authority have been reversed. The "doctrine" of the Courts had been that "any degree of mental unsoundness, however slight and however unconnected with the testamentary disposition in question, must be held fatal to the testamentary capacity of a testator," and this doctrine, Chief Justice Cockburn said, had been very emphatically declared by Lord Chancellor Brougham, presiding over the Judicial Committee of the Privy Council in Waring *v.* Waring. The head-note to this remarkable case, 6 Moore, P. C. Rep., thus explains the purport of the judgment:—

"This exposition of the doctrine of monomania and partial insanity, as applied to wills, was that if the mind is unsound on one subject, providing that unsoundness is at all times existing on that subject, it is erroneous to suppose such a mind is really sound on other subjects; it is only sound in appearance, for if the subject of the delusions be presented to it,

the unsoundness would be manifested by such a person believing in the suggestions of fancy as if they were realities, any act therefore done by such a person, however apparently rational that act may be, is void, as it is the act of a morbid or unsound mind."

"This doctrine," said Cockburn, "was, perhaps, more fully expressed by Lord Penzance in Smith *v.* Tebbitt." Thus: "A person who is affected by monomania, although sensible and prudent on subjects and occasions other than those upon which his infirmity is commonly displayed, is not in law capable of making a will. . . . For I conceive the decided cases to have established this proposition, that if disease be once shown to exist in the mind of a testator, it matters not that the disease be discoverable only when the mind is addressed to a certain subject to the exclusion of all others; the testator must be pronounced incapable. Further, the same result follows, though the particular subject upon which the disease is manifested have no connection whatever with the testamentary disposition before the Court."

This legal doctrine was abolished as wrong, and a contrary rule substituted in its stead by the judgment of Chief Justice Cockburn, presiding in Banco of Queen's Bench, Justices Blackburn, Mellor, and Hannen concurring therein, upon the motion for a new trial in Banks *v.* Goodfellow, July 6, 1870. The testator John Banks was admitted to have been of unsound mind; he had been confined in an asylum and remained subject to certain fixed delusions. He had conceived a violent aversion to a man long since dead, whom he believed still to pursue and molest him, and the mention of this man's name threw him into violent excitement. He frequently believed that he was pursued and molested by devils or spirits, whom he believed to be visibly present. There was conflicting evidence as to his general insanity, but "from September [the will being made on December 2 following] 1863, he had a succession of epileptic fits, and a blister was applied to his head, and the medical man who attended him throughout this period deposed that his mental power, such as it was, suffered from these fits, and that he considered the testator insane and incapable of transacting business during the whole time." On

the other hand, it appeared that he managed his limited affairs and was careful of his money. The Westmoreland jury found for the validity of the will, which in-so-far as it bequeathed his property to his niece who lived with him was a natural disposition of his property; but, inasmuch as his niece was his heir-at-law and would have inherited without the will, it was a vain and unnecessary one. The niece died shortly after the testator, and her heir, being no kin to the testator, claimed under the will, thus disinheriting the testator's nephew, the plaintiff, who strove to cancel the will on the ground of the incapacity of the testator.

From this statement, somewhat abbreviated from that made by the Chief Justice himself, it will appear to any practical psychologist, that in order to reduce the issue to a question of principle, extremely large assumptions were made in the judgment, in that it assumed the absence of general insanity, and also that "the delusions must be taken neither to have had any influence on the provisions of the will, nor to have been capable of having any." Thus trimmed to the most convenient condition for argument, of course the question became "whether a delusion, thus wholly innocuous in its results as regards the disposition of the will, is to be held to have had the effect of destroying the capacity to make one." The simple dogma of the older jurists that an insane person was incapable of making a testament "*quia mente caret*" is obviously unsatisfactory in the opinion of the Chief Justice, "when the fact becomes recognised that a man may labour under *harmless delusions which leave the other faculties of his mind unaffected*, and leave him free to make a disposition of his property uninfluenced by their existence." The judgment, therefore, with regard to direction was that "a jury should be told in such a case that *the existence of a delusion compatible with the retention of the powers and faculties of the mind will not be sufficient to overthrow the will, unless it were such as was calculated* to influence the testator in making it;" and, looking at the evidence by the light of this doctrine, the Court refused a new trial, on the ground that "in our judgment the only proper or possible result must be a second verdict establishing the will."

In order that we may the better appreciate Chief Justice Cockburn's argument and the judgment founded upon it, the judgment in Waring *v.* Waring delivered by Lord Chancellor Brougham, with the concurrence of Lords Langdale, Lushington and Pemberton Leigh, of the Judicial Committee of the Privy Council, ought to be considered; seeing that it is the faultiness of this opposite view, of which this decision is the most remarkable and authoritative expression which is believed to have justified its reversal. Lord Brougham said that, in speaking of disease or decay affecting the mental faculties being more or less general, or of affecting more or fewer of these faculties, "we must always keep in view that which the inaccuracy of ordinary language induces us to forget, that *the mind is one and indivisible,* and that when we speak of its different powers or faculties, as memory, imagination, consciousness, we speak metaphorically, likening the mind to the body, as if it had members or compartments, whereas, in all accuracy of speech, we mean to speak of the mind acting variously, that is remembering, fancying, reflecting, the same mind in all these operations being the agent."

A man's mind being one, Lord Brougham says that cases of monomania are incorrectly called partial insanity. "We are wrong in speaking of partial unsoundness, we are less incorrect in speaking of occasional unsoundness; we should say that the unsoundness always exists, but it requires a reference to the peculiar topic, else it lurks and appears not. But this malady is there, and the mind is one and the same; it is really diseased while apparently sound, and really its acts, whatever appearances they may put on, are only the acts of a morbid and unsound mind." "We never can rely on such acts, however rational in appearance, because we have no security that the lurking delusion, the real unsoundness, does not mingle itself with or occasion the act." This latter point, not in any way dependent upon metaphysical opinions or doctrines, will be most readily admitted by those who have the largest experience of the insane. One can never tell when their insanity will not crop out; so that their testamentary and other acts, which may appear to be quite reasonable, may have an insane motive, and indeed are rarely altogether

free from some admixture of such motive; and Brougham's observation is perfectly justified that "it is hardly possible that any will can be so framed as to rebut all presumptions of insanity arising from proved facts," for all the conduct of the insane is tainted with at least suspicion of insane motive; nay, the presumption is and always must be that there is some degree of insane motive in all their conduct, or, in other words, that the motive of an insane man never is quite what it would have been had he been of sound mind. Not that the motive is altogether perverted and different, but that, looking to the full composition of motive in memory, fancy, feeling and desire, and all the other faculties which go to influence conduct, the motive of the most typical monomaniac, whether it be determined to call him insane as to parts of his mind, or insane as to occasions of thought, or insane as to a limited range of things or affairs, cannot be relied upon as a sane motive, even if the act be apparently that of a sane man.

On this point Lord Brougham's opinion is inexpugnable, but not so in regard to his objection to the term "partial insanity," on the ground that there is no such thing, seeing that the mind being one, all insanity must be held to affect the whole mind. It is much more true that all insanity is partial insanity, and that there is no such thing as insanity which is not partial. As the existence of bodily disease implies the continuance of life, so the existence of insanity implies the continuance of mind. Therefore coma, and perhaps the extreme of amentia, can scarcely be called insanity, which is perversion or defect of mental activity, not its abrogation. But partial insanity, as it was understood and objected to by Brougham, was not this kind of partiality. It was the partiality with which we speak of local disease of the body in contradistinction to constitutional or general disease, and his objection to it on the ground that mind is one and indivisible, and therefore that its affections cannot be limited to a part or parts, although an argument of the metaphysical kind, is still one which can scarcely be avoided in thorough discussion of such a question. Indeed it would seem that we are somewhat less metaphysical, and more in accordance with the common sense and opinion of

mankind, when we say with Brougham that mind is one thing, and that this habit of speaking of its various activities as if they were distinct things is but a metaphorical mode of expression due to the imperfection of language, than if we were to adopt Cockburn's view of the independence of the faculties.

In the Banks *v.* Goodfellow judgment, Chief Justice Cockburn "did not think it necessary to consider the position assumed in Waring *v.* Waring, that the mind is one and indivisible, or to discuss the subject as matter of metaphysical or psychological inquiry. It is not given to man to fathom the mystery of the human intelligence, or to ascertain the constitution of our sentient and intellectual being." And yet he immediately proceeded to discuss the question in a metaphysical or rather psychological manner as follows:—"Whatever the essence of it may be, every one must be conscious that the faculties and functions of the mind are various and distinct, as are the powers and functions of our physical organisation. The senses, the instincts, the affections, the passions, the moral qualities, the will, perception, thought, reason, imagination, memory, are so many distinct faculties or functions of the mind. The pathology of insanity and the experience of its various forms teach us that while, on the one hand, all the faculties, moral and intellectual, may be involved in one common ruin, as in the case of the raving maniac, in other instances one or more only of these faculties or functions may be disordered, while the rest are left unimpaired and undisturbed; that while the mind may be overpowered by delusions which utterly demoralize it and unfit it for the perception of the true nature of surrounding things, or for the discharge of the common obligations of life, there often are, on the other hand, delusions which, though the offspring of mental disease, and so far constituting insanity, yet leave the individual in all other respects rational, and capable of transacting the ordinary affairs and fulfilling the duties and obligations incidental to the various relations of life. No doubt when delusions exist which have no foundation in reality and spring only from a diseased and morbid condition of the mind, to that extent the mind must necessarily be taken to be unsound; just as the body, if any of its parts or functions is affected by local disease, may be said to be unsound, though

all its other members may be healthy and their powers or functions unimpaired. But the question still remains whether such partial unsoundness of the mind, if it leaves the affections, the moral sense, and the general power of the understanding unaffected, and is wholly unconnected with the testamentary disposition, should have the effect of taking away the testamentary capacity."

Further on he declares that the fact must be recognised that "a man may labour under harmless delusions, which leave the other faculties of his mind unaffected, and leave him free to make a disposition of his property, uninfluenced by their existence." Surely the above quotation will justify us, not only in declaring that it is impossible to discuss such subjects except as matters of psychological inquiry, but that the Chief Justice did so discuss them, and did not discuss them well, in consequence of his abortive attempt to assimilate mind more closely than in the nature of things will bear to be done to the functions and even to the parts of the body. There is no real resemblance or community of nature between a delusion and a part of the body affected by local disease; neither is it true that a delusion is a faculty of the mind, as Cockburn appears to think when he speaks of delusions and other faculties of the mind. The argument which compares a delusion to an injured hand, or an eye which a man can pluck out and cast from him, needs no refuting; but to consider a delusion one of the mental faculties is a serious error, inasmuch as it seems to vitiate the whole of an argument in many respects acute, able, and learned. It is, in his own forcible language, "a position obviously unsatisfactory when the fact becomes recognised" that a delusion is a state of mind in which all the mental faculties or activities are affected. Sense, perception, judgment, feeling, and whatever else goes to constitute mind are, and must be, each and all affected in such a fundamental perversion of the reason. In the concrete instance discussed, John Banks, the testator, who had conceived a violent aversion to Featherstone Alexander, and, notwithstanding the death of the latter, believed that he still pursued and molested him, and the mention of whose name threw him into violent excitement, and who believed that he was pursued and molested by devils who were visibly present to

him, must have had perversion of the sense and perception, constituting hallucination; must have had the faculty of comparison perverted, or he would have recognised the false sense, as Nicholai and others have done; must have had his feeling perverted, or he would not have been thrown into violent excitement by the chimera of persecution by a man long since dead, but whose death he could not remember. The mental activities implicated in these beliefs were as little partial, as regards the field of mind which they implicated, as the mental activities of the Chief Justice himself, in the judgment which he delivered upon them, or as those of the Judges whose opposite judgments he superseded. It is a marvellous thing that a man can be stark staring mad upon one train of ideas, and that you can start the manifestation of his madness by a word, while upon all other topics and trains of thought he is comparatively rational and seemingly of sound mind. But it is of the first importance to discussions on the influence of delusions, either on responsibility or on testamentary capacity, to be assured that not part of the mind but the whole of a man's mind, whatever we may think of its unity or its composition, is affected by such a delusion, which absorbs the man while he is excited by it; not one of his faculties to the exclusion of others, but the whole of his faculties on the particular subject or subjects respecting which he entertains insane delusions. We cannot but think that Chief Justice Cockburn misapprehended and therefore misstated the argument of Lord Brougham in Waring *v.* Waring, and that of Lord Penzance in Smith *v.* Tebbitt, since he says that the doctrine for the first time laid down by them "may be shortly stated thus. To constitute testamentary capacity, soundness of mind is indispensably necessary. But the mind, though it has various faculties, is one and indivisible. If it is disordered in any one of those faculties, if it labours under any delusion arising from any such disorder, though its other faculties and functions may remain undisturbed, it cannot be said to be sound. Such a mind is unsound, and testamentary incapacity is the necessary consequence." The doctrine referred to really appears to have been so much opposite of this, that it would be far more correct to state it thus. The mind being one and

indivisible cannot be disordered in any one of its faculties; and mental disease implies disorder of all. Insane delusion affects all the mental faculties, although such affection may be latent when the subject of delusion is not before the mind. Such a mind is unsound, and testamentary incapacity is the necessary consequence. It was not argued by Lords Brougham and Penzance that insane delusions on a limited subject of belief and feeling must necessarily affect the motives of a man making a will, but it was argued that insane delusions would cast such a doubt on those motives, and would moreover throw such a taint of suspicion upon the soundness of the understanding of the person so affected as to amount to testamentary incapacity. Chief Justice Cockburn, on the other hand, assumed as the foundation of his argument that insane delusions might exist, and in the case under consideration did exist, "delusions which had, in point of fact, no influence whatever on the testamentary disposition in question," "delusions which had not, nor were calculated to have any influence on him in the disposal of his property;" "and the question is whether a delusion thus innocuous in its results as regards the disposition of the will, is to be held to have the effect of destroying the capacity to make one."

If this line of argument is not begging the question it is very like that common fallacy, for if it be admitted that the delusion must be taken neither to have had any influence on the provisions of the will, nor to have been capable of having any, *cadit quæstio*. But if such admission be not made, as "in this case we are dealing with," it certainly ought not to have been made, it is the argument which fails. "In this case we are dealing with," the delusions, as described by the Chief Justice, might have influenced the testator's disposition of his property in an infinite variety of ways. Take one of the most probable, namely that he was so preoccupied by his delusions that he did not recollect the existence or the claims of the nephew, who was not present, but only those of the niece, who was present, for it is a frequent effect of delusion to exclude reflection upon subjects not within its range. And again, in considering "this case we are dealing with," what is more probable, nay, almost certain, than that the only relative

living with a person labouring under such delusions of persecution by a dead man, and by devils and evil spirits, would become intimately related with those delusions, although they did not directly include or refer to herself? Would she not have had to comfort him under his fanciful terrors, to assume the *rôle* of protector against his persecutors, and would such a position have no effect upon the dispositions of the lunatic? Clearly this reasonable probability was quite opposite to that which the Chief Justice so resolutely assumed as the certainty, for he did not put it as a probability but as a certainty, that the delusions could have had no influence whatever on the testamentary disposition.

We venture therefore to think that the argument of Chief Justice Cockburn did not quite meet, and that certainly it did not entirely answer, the argument of Lords Brougham and Penzance with regard to the influence of delusion on testamentary capacity. At the same time we cannot fully concur in the conclusions of Lords Brougham and Penzance. They may follow the psychological premises, but they are too formal and definite to adapt themselves to the infinite varieties of Nature, even as she is seen in the aberrations of the human mind. Perhaps less injustice would be done under their rule of exclusion than by Chief Justice Cockburn's rule of admitting the wills of lunatics with delusion, for without doubt a great and increasing amount of injustice is done every year by the probate of the wills of lunatics which ought never to have been made. It is a great social privilege, that of bequeathing one's property to whomsoever or for whatsoever one thinks fit, possessed in its unfettered condition by the English race almost alone among the nations, and no one can peruse the eloquent remarks which the Chief Justice made in this judgment upon the moral responsibility of exercising this privilege, or rather of discharging this duty aright, without an increased feeling that its abuse ought to be strictly guarded. At the present time the legal doctrine as to testamentary capacity and incapacity does not appear to be in a satisfactory state; seeing that the old doctrine, accepted for a long period and reaffirmed perhaps upon insufficient argument, has been upset, and an opposite doctrine substituted upon argument far from satisfying and

conclusive. It may at least be objected to all the argument on both sides that far too much stress has been laid upon delusion, as if it were the main element of insanity, and as if it were always very much the same kind of thing in nature and degree, only applied to different kinds of subjects. With all of those great Judges a delusion is a very definite and positive state of belief. It is wrong belief; but it is so strong that a man who entertains it is sure to act upon it. But, we will not say with Cockburn, "those who are most conversant with the pathology of mental disease," but use the better term with which he follows up that too flattering phrase, "those who have most experience of insanity in its various forms," well know that insane beliefs are like sane beliefs, in all their infinite variety of strength or weakness, clearness or obscurity, persistency or changeableness, activity or latency, power or incapacity to influence action; and that they have, in almost as great a variety as sane beliefs, connection with or independence of an infinite variety of passions or emotions, and of intellectual conditions other than beliefs. A man may possibly have a delusion which would seem extremely likely to influence his will, and yet from some quality in the nature of his belief in his delusion, it shall not influence his will or seriously affect his conduct in life. On the other hand, another man may well have a delusion which Chief Justice Cockburn would have been too ready to say "was wholly innocuous as regards the disposition of his will," and yet, through one of those cross-cuts of perversity so rife in the insane mind, the will shall be nothing but the expression of the delusion. These facts, which will be in agreement with the experience of all competent observers, point to the conclusion that testamentary incapacity ought to be decided upon, not by the existence of delusions or their supposed interference or non-interference with the reasonings or feelings of the testator, but upon all the circumstances of each individual case, which is the identical doctrine advocated by Cockburn himself with regard to the influence of delusion in determining irresponsibility for criminal acts.

But in this judgment the Chief Justice went beyond this reasonable doctrine, when he so far depreciated the importance of delusion as to declare that the opinion must be looked upon as

merely speculative, and unsupported by proof, which assumed the probability that where insane delusion has shown itself, a greater degree of mental unsoundness exists than has actually become manifest. It surely is not a matter of merely speculative opinion, but one rather of pretty constant experience, that where fixed delusions exist the mind is profoundly affected. In this very case "the mere mention of Featherstone Alexander's name [the dead man who still pursued and molested him] was sufficient to throw him into a state of violent excitement." "But as the delusion was not manifested at the time of making the will, it is a question whether the delusion was not latent in the mind of the testator." Just so. There was a greater degree of mental unsoundness than was actually manifest. There was delusion and a morbid state of temper and feeling which a word could at any hour explode into violent excitement.

But it must be admitted that some delusions do not indicate any greater degree of mental unsoundness than that which is always manifest, and that there are some delusions which may be predicated as unlikely, though not incapable, of having any effect upon a will. The varying delusions of hypochondriasis are quite different from the fixed delusions of persecutions such as were entertained by Thomas Banks; and while these would not necessarily indicate any mental unsoundness beyond the unfounded and extravagant opinions they represent, the others would necessarily indicate profound mental lesions which would be almost certain to manifest themselves in aberrations and defects of the understanding, and in perversions of the affections on other subjects than those immediately referable to or connected with the delusions themselves. Mr. Justice Stephen, than whom no one has investigated this difficult corner of law with more philosophical and diligent study, made the following remark as to the unexpected and incalculable influence of insane delusion, in his evidence before the Select Committee on the Homicide Law Amendment Bill:

"Where you have a specific delusion of that kind it shows that the mind itself is so deeply disordered in all kinds of ways, you cannot draw the inference that there was an intention to kill or to do grievous bodily harm from the fact of killing as you

could in other cases:—when you get a man under any definite delusion whatever, for aught you can tell the workings of his mind may be such that the act which to you appears to be murder, appears to him in quite another light; and if you read books which give accounts of the workings of the minds of mad people, you will find that directly you get a definite delusion set up, the process of the mind is vitiated as well as the mere result. The delusion runs through everything."

This undoubtedly is true of most delusions, and would especially be true of fixed delusions of persecution accompanied by hallucinations of sense and associated with epilepsy, such delusions, in fact, as those of Thomas Banks, which, in the opinion of the Court of Queen's Bench, left the intelligence and the emotions so free and unaffected. But there are delusions and delusions; delusions in the nascent and others in convalescing conditions of mental disease; delusions which indicate "deep disorder of the mind in all kinds of ways," so that "the process of the mind is vitiated as well as the result;" and contrasted with them there are delusions which scarcely affect the mind beyond their own range, and which, weak and changeable, cannot have the import attached to them in the above quotation. And it is worthy of remark that the Judges sometimes employ this term "delusion" to indicate disturbance of thought arising from insanity, without any particular belief in imaginary facts: thus Lord Penzance, in his judgment, Smith *v.* Tebbitt: "It is no doubt true that mental disease is always accompanied by the exhibition of thoughts and ideas that are false and unfounded, and may properly be called 'delusive.' But the question of insanity and the question of 'delusions' is really one and the same. The *only* delusions which prove insanity are *insane delusions*—and the broad inquiry into mental health or disease cannot in all cases be either narrowed or determined by any previous or substituted inquiry into the existence of what are called delusions."

Chief Justice Cockburn, however, did not adopt this loose interpretation of a term, whose definite meaning it is of the utmost importance to fix with all possible exactness. We have seen that in his attack on the Hopwood will he admitted that there was no evidence of delusion, although there was abun-

dant evidence of loss of memory and of confusion of thought, extending to complete testamentary incapacity. Yet Mr. Hopwood had ideas respecting his eldest son, whom he had disinherited, which were "false and unfounded," and might therefore, according to Lord Penzance, be called "delusive." If Lord Penzance had turned his dictum the other way first, and said that the only delusions of value as evidence being insane delusions, to prove the existence of such delusions it is needful first to prove the existence of insanity, it would have been more logical, although such delusions might have included those beliefs of the insane, which may by possibility be entertained by ignorant, flighty, and fanciful people who are not insane. Insane delusions, however, are recognised by their accompaniments,—insane history, insane conduct, insane feeling, insane physical symptoms,—and by their character, exhibited by comparing them with like beliefs, with like accompaniments, in other persons who are unquestionably insane, that is to say, sufficiently like to convince a candid and instructed mind that they are essentially alike.

But these reflections, if they are well-founded, tend greatly to weaken the position of the Lord Chief Justice with regard to the possibility of such delusions as those of Thomas Banks existing without "the mind becoming deeply disordered in all kinds of ways, so that you cannot draw any inference as to intention," even so simple an inference as to whether he intended to kill a man or not; more so as to an intention even more complex regarding testamentary dispositions. For, observe, these delusions had all the accompaniments of insanity which we have indicated. There was an insane history, residence in the county lunatic asylum; insane conduct, violent excitement; insane feeling, violent aversion; insane physical symptoms, epileptic fits; and it will be scarcely denied by any candid person conversant with lunatics that these beliefs and their accompaniments resemble the insane beliefs and the other marks of insanity in thousands of persons suffering from general insanity so closely that there could be no possibility of doubting that they were of the same kind and nature.

The Lord Chief Justice proceeds in his judgment to comment with admirable precision and elegance of diction upon the great

moral responsibility involved in the discharge of that duty so largely committed to the individual in this country of disposing of his property after death to whom he will. He recognises the power of mental disease to poison the affections and to pervert the sense of right, and thus to abolish the responsibility which this duty involves; but here again he intervenes to assert the possibility of delusion neither exercising, nor being calculated to exercise, any influence upon the particular disposition of the testator. The great moral responsibility attached to the duty of making a just and righteous will appears to us to increase rather than to weaken the argument in favour of the old rule of law which denied the exercise of so responsible a privilege to any one proved to be of unsound mind.

From this aspect of the question the Chief Justice proceeds to discuss the effect of enfeeblement of memory and defect of mind upon testamentary capacity. On this question the most important judgment he quotes is that delivered by Erskine, for the Judicial Committee of the Privy Council, in Harwood *v.* Baker [3 Moore, P.C.], viz.: "Their Lordships are of opinion that in order to constitute a sound disposing mind, a testator must not only be able to understand that he is by his will giving the whole of his property to one object of his regard, but he must also have the capacity to comprehend the extent of his property, and the nature of the claims of others whom by his will he is excluding from participation in that property," &c., and the Chief Justice asks, "why should not this standard be also applicable to mental unsoundness produced by mental disease? It may be said that the analogy between the two cases is imperfect; that there is an essential difference between unsoundness of mind arising from congenital defect or supervening infirmity, and the perversion of thought and feeling produced by mental disease, the latter being far more likely to give rise to an inofficious will than mere deficiency of mental power. This is no doubt true, but it becomes immaterial on the *hypothesis* that the disorder of the mind has left the faculties, on which the proper exercise of the testamentary power depends, unaffected; and that a rational will, uninfluenced by the mental disorder, has been the result."

It is indeed an hypothesis that disorder of the mind as distinguished from "congenital defect or supervening infirmity can leave those faculties unaffected upon which the proper exercise of the testamentary power depends." What are these important and efficient mental faculties which can thus be left unaffected by insanity? The supposition, even as an hypothesis, is that which we conceive to be the fundamental error of the whole judgment, namely the metaphysical or psychological conception that a man's mind is merely a bundle of diverse faculties more or less independent of each other, some of which may be affected by insanity, while others upon which the proper exercise of the testamentary power depends, remain unaffected. The Chief Justice does not adduce any evidence in support of his conviction upon which the judgment and the new law depends, namely that a man's mind is thus constructed; but while disclaiming metaphysical inquiry with great emphasis, he adduces the most metaphysical of arguments, namely that of consciousness, in support of his views. "Every one must be conscious that the faculties and functions of the mind are various and distinct, as are the powers and functions of our physical organisation." Our only possible reply is that we have no such consciousness, but that on the contrary we are conscious that mind in us is one thing or power with various modes or directions of activity, but with one indecomposite individuality. And we think that the neurological science already acquired and even that which we may aspire to, from the pursuit of the same methods which have won all recent discovery, indicate the oneness of mind as contradistinguished from the localisation and diversity of brain function. In neurology we cannot get beyond or behind impressions, reflections, and recollections of sense, and as often as we venture to distinguish a bit of mind apart from a man's whole mind, it will be found to be merely a bit of sense felt, reflected or recollected. But a man's whole mind, that is to say, in the words of this judgment, "the senses, the instincts, the affections, the passions, the moral qualities, the will, perception, thought, reason, imagination, memory," these are not "so many distinct faculties or functions of the mind," but the tissue of sense-impressions, inherited and acquired,

inextricably woven into one whole state of consciousness which attains to new powers or activities, catalogued under the impressive but often misleading terms above quoted. In conclusion, we are of opinion that the late Chief Justice of the Queen's Bench and the great Judges over whom he presided were right in challenging the extremely narrow legal rule with regard to testamentary incapacity, which they reversed, but that the grounds upon which they acted were fallacious, the argument wrong and misleading, the new rule dangerously lax and wide, and the result in the particular instance to which it was first applied a miscarriage of justice. In the early days of the old rule, when an insane man was said to be unable to make a will simply *quia mente caret*, insanity covered but a very moderate portion of the wide field over which it has since been extended. An insane man then almost invariably indicated a person about whose testamentary incapacity there could be no question. But when the same strict rule came to be applied to new forms and degrees of insanity, it was inapplicable, and indeed it then became that very fallacy which we have indicated as the error of those who argue that every insane man ought to be exempt from responsibility for crime; the fallacy of arguing *a dicto secundum quid ad dictum simpliciter*.

There being in all forms of insanity infinite degrees of mildness and severity, it became obviously unjust to apply that rule to the mild and slight degrees of mental disease and defect which was only applicable to degrees of greater intensity. The question of course was always essentially one of motive, that is to say ethical, or, if you like, psychological; but concurrently there was one, we will not say for the medical man, but for any man who diligently studies all the mental qualities and conditions of the human being. This the authors of the old legal rule could not do, because degrees of insanity were scarcely recognised in those days. This also the authors of the new rule have failed to do, because they adopted a metaphysical conception which misled them. What legal rule may eventually be devised and upheld, which shall delineate fairly and fully the characters and degrees of mental diseases which shall, and those which shall not, carry with them testamentary incapacity, must depend upon the attainment

of a better knowledge both legal and mental, and of a more intimate concurrence between equity and science. Whatever the rule may be, however, its application must not unfrequently "involve considerable difficulty and require much nicety of discrimination," as the Chief Justice admitted of inquiries under his own rule. The manner, however, in which such difficulties may be overcome will surely not be by metaphysical or psychological discussion, but by the painstaking method of comparison described by Lord Penzance [Smith *v.* Tebbitt], first, by the comparison which common men make of the words and deeds, as indicating the thoughts of the testator, with the standard of sanity they bear in their own minds; and secondly, the more instructive comparison which those who are conversant with the insane can make between the sayings and doings of the testator with the sayings and doings of those who are undoubtedly insane to an intestamentary degree. And it is, as Lord Penzance argues, by this double comparison of the mind of the testator with the sane mind of the Court, and with the insane mind as it is known and described by competent observers, by which that nicety of discrimination which these inquiries demand may be attained.

We have been led into a longer criticism of this famous judgment than we had intended or expected. Its intrinsic importance, which cannot easily be overestimated, conjoined with the profound respect we entertain for the memory of its author, has forbidden us from expressing our objections with less consideration and amplitude. This judgment in Banks *v.* Goodfellow, full of learning and research as it is, covers much of the ground of insanity in its civil relations, and indeed the Chief Justice himself informed the writer of these pages that he considered this judgment as the full expression of his opinions upon the whole subject. That these opinions are original, and constitute an entirely new law upon the subject, is a sufficient justification, if one were necessary, for subjecting them to a free criticism, which, so far as we know, has not before been done.

THE DIFFERENTIAL DIAGNOSIS OF PARALYSIS.[1]

BY BYROM BRAMWELL, M.D., F.R.C.P. EDIN.

Lecturer on the Principles and Practice of Medicine, and on Practical Medicine and Medical Diagnosis in the Extra-Academical School of Medicine, Edinburgh. Late Physician and Pathologist to the Newcastle-on-Tyne Infirmary, &c. &c.

PARALYSIS is a symptom, not a disease. In every case, therefore, in which it comes before us, we must endeavour to ascertain its cause.

The following are the steps in the inquiry:—

1. Is the paralysis genuine?
2. If genuine, is it functional or organic?
3. If organic, what is the position, extent, and pathological character of the lesion?

Step No. 1.—*Is the paralysis genuine?*

The great characteristic of genuine paralysis is "defective or abolished muscular contraction, *while an effort of the will remains.*" (Erb.) In feigned (sham) paralysis there is defective or abolished muscular contraction, because the supposed patient *does not choose to make* an effort of the will. The recognition of feigned paralysis, when the impostor is well up to his trade, may be a matter of great difficulty. The chief points on which reliance is to be placed in making the diagnosis are:

A. *Negative Evidence.*—1. The fact that there are no signs nor symptoms of organic disease.

Feigned paralysis *is to be suspected* when:

[1] A Lecture delivered in the Extra-Academical School of Medicine, Edinburgh, during the summer session 1880, and read as a paper before the Medico-Chirurgical Society of Edinburgh, January 5th, 1881.

(*a*) The condition of the muscles, as regards their nutrition, irritability (mechanical and electrical), tonicity, the condition of the reflexes, &c., is quite normal.

(*b*) There are no associated nerve symptoms; particularly, (1) No symptoms of hysteria, in which condition paralysis of a functional character often occurs. (2) When there is no evidence of mental derangement. (In some cases of mental disease the patient does not or will not move a limb or limbs, but in such cases there is no intention to deceive; the paralysis is not therefore *feigned* in the sense in which I have used the term.)

(*c*) There are none of the conditions present which predispose to or give rise to paralysis, such as disease of the heart, arteries, kidneys.[1]

B. *Positive Evidence.*—If, in addition to the negative facts already mentioned, there is any positive evidence in favour of imposture, *then and then only* is a diagnosis of feigned paralysis justifiable.

The positive facts are:

1. The presence of any anomalous symptoms, such as:

(*a*) Irregular distribution of the paralysis. In cases of hemiplegia, the fact that the arm suffers less than the leg,[2] or that the arm on one side and the leg on the other are affected.

(*b*) Any peculiarity in the paralysed parts. The circumstance that in walking the hemiplegia patient does not circumduct the leg, though not absolute, is of some weight.[3]

[1] Impostors may of course have heart, kidney, or arterial disease. The converse proposition, therefore, that all persons who have heart, kidney, or arterial disease, and who manifest symptoms of paralysis, are impostors, is obviously not true.

[2] In some genuine cases the arm suffers less than the leg, but this is so rare that, if taken in conjunction with the negative facts, mentioned above, it would be very strong evidence indeed in favour of imposture.

[3] In some cases of genuine organic paralysis there is no circumduction of the leg. In functional cases, too, the leg is not circumducted; indeed, according to Todd, this is one of the points of distinction between organic and hysterical hemiplegia.

The fact mentioned by Todd that, in cases of feigned paralysis, when the patient is told to stoop forwards and pick up something from the ground, the paralysed arm is kept fixed to the side instead of falling forwards of its own weight would be conclusive. (In cases of genuine paralysis the arm may be kept fixed to the side by rigidity [contracture]; but in such cases the other facts, especially the condition of the deep reflexes, are so distinctive as to be quite unmistakable.)

2. Any peculiarity in the history, mode of onset of the attack, &c. The fact, for example, that the paralysis followed a railway collision, if coupled with negative evidence, is of some weight in favour of imposture.

3. The fact that the patient has something to gain by the imposture. That he is claiming damages from a railway company; that he has no fixed abode, that he has been in and out of many hospitals, that he is, in short, a "hospital bird" trying to keep a roof over his head by his deceit.

Simulated, but not sham, paralysis.

Paralysis may be simulated when there is no intention on the part of the patient to deceive, as in:

(1) Those cases of mental disease already referred to (see page 28). Under this head we may perhaps include some cases of hysterical paralysis.

(2) In cases of insensibility (coma), it is sometimes very difficult to say whether the patient is paralysed or not.

In the great majority of cases of coma if there is any paralysis it is unilateral (hemiplegia). In such cases a careful comparison of the muscles on the two sides of the body is to be made, as regards:

(*a*) *The condition of the reflexes.*—In cerebral paralysis (hemiplegia) the superficial reflexes are abolished, and the deep reflexes increased on the paralysed side. (The increase of the deep reflexes is usually not apparent until the stage of coma passes off.)

(*b*) *The tonicity of the muscles.*—The paralysed muscles are usually softer and more relaxed, fall with a dead weight, &c. In the case of the face the paralysed cheek is blown

out during expiration, saliva flows from the angle of the mouth on the paralysed side, &c. In the face, too, where the symmetry of the features depends upon the equality of action of two opposing sets of muscles, we can usually detect some drawing of the features to the sound (opposite) side. The presence of (paralytic) conjugate deviation of the eyes and head would at once, of course, settle the question, for this symptom only occurs in severe cases.

(*c*) The temperature, degree of moisture, colour of the skin, on the two sides. In many cases of hemiplegia alterations in the vaso-motor condition of the paralysed parts are observed. In the earlier stages the temperature on the paralysed side is usually higher, the skin more moist, and the colour deeper, than on the sound side.

(*d*) As regards spontaneous movements.

In many cases of incomplete coma, the patient does make some spontaneous movements, but they are limited to the sound side. (An exception to this statement occurs in the so-called "associated movements," which may occur on the paralysed side. These associated movements are usually observed after the coma has disappeared.)

The fact that *convulsive movements* are limited to one side does not, as is sometimes erroneously thought and stated, prove that the other (the immobile) side is paralysed. In fact, in the great majority of cases, the contrary holds good. In the large majority of cases of unilateral convulsions we should be correct in predicting that, if either side were paralysed, it would be the same (i.e. the convulsed) side.

Paralysis may also be simulated by other genuine affections.

1. *Disorders of co-ordination*, notably Locomotor Ataxy, were, and are, even still, mistaken for paralysis. That the disturbance of motion in such cases (the difficulty of walking in the case of locomotor ataxy) is not due to paralysis is proved by the fact that, the gross force of the muscles is well preserved; in some cases, indeed, in which the muscular development is great, the gross force is much above the normal average.

2. *Painful affections.*—In some cases of rheumatic fever, for example, the parts are instinctively kept as quiet as possible, and such cases may be mistaken for paralysis.[1]

Step No. 2.—The paralysis is genuine.—Is it functional or organic?

By functional paralysis we mean those cases in which the loss of power depends upon changes in the motor nerve-tissue of so slight a character as to baffle all our present means of investigation, in contradistinction to those cases of paralysis which depend upon an obvious organic cause.

Functional paralyses are usually temporary in character; the paralysis often sets in suddenly and as quickly disappears.

Under the head of functional paralyses are included:—hysterical paralysis, epileptiform paralysis, paralysis from idea, malarial paralysis, reflex paralysis, and some others.

Hysterical Paralysis.

Before entering upon the differential diagnosis of hysterical paralysis, it is necessary to insist upon the fact, that all cases of paralysis occurring in hysterical patients are not necessarily hysterical (functional). Hysteria, indeed, is frequently associated with organic disease of the nervous system. The presence of hysteria, or a history of hysterical fits, is only, at the most, corroborative evidence. A diagnosis of hysterical paralysis, even when the patient is hysterical, should never, then, be made unless the most scrupulous examination has failed to detect the signs and symptoms of organic disease.[2]

The differential diagnosis of hysterical paralysis is sometimes very difficult, and, since it is a matter of the first importance, the points on which reliance is to be placed in solving the problem will be given in some detail.

A. *Negative Evidence.*

1. The fact that there is nothing in the condition of the

[1] It must be remembered that in some of these rheumatic cases a true paralysis does occur.

[2] This of course raises the question as to what is positive evidence of organic disease—a question which is not yet in every respect absolutely settled. (See remarks on the ankle-clonus, page 32.)

paralysed muscles distinctive of organic paralysis (no "reaction of degeneration," no "rapid atrophy").

In cases of hysterical paralysis the nutrition of the muscles is usually well preserved. This is certainly the fact in the earlier stages; in the latter period of long-continued cases there is atrophy, but it is not more than can be accounted for by disease.

The *irritability* (mechanical and electrical) of the muscles is normal, or possibly slightly increased, but (in the earlier stages) not markedly decreased.

The *tonicity* of the muscles is normal or increased. Sometimes there is rigidity (a spastic condition). In the later stages of chronic cases the muscles may be flaccid.

The condition of the reflexes.—In two cases which I have recently had an opportunity of examining, the skin reflexes were abolished, while the deep reflexes were increased. In both of these cases the ankle-clonus was well marked. On this point, therefore, I differ from Dr. Gowers, who, in his recent most admirable book on the diagnosis of the spinal cord, states that, the presence of the ankle-clonus is proof positive of organic disease. Professor Charcot and Dr. Buzzard have also observed the ankle-clonus in hysterical paralysis.

It will be gathered from this description that, in my opinion, the condition of the muscles in hysterical paralysis (hysterical hemiplegia) differs in no way from the condition of the muscles in many cases of organic cerebral paralysis. The presence of "rapid atrophy" and the "reaction of degeneration," would, of course, prove that the paralysis was organic, and that it was due to a lesion of the nerve trunk or nerve nucleus.

2. That there is no evidence of such disease of the nervous system as might cause, or as is usually associated with, organic paralysis. A careful and detailed examination of the other parts of the nervous system must, therefore, be made in cases of supposed hysterical, as indeed in all cases of paralysis.

It is specially important to note the condition of the optic discs. In some cases of paralysis and hemi-anæsthesia, the

conditions of the optic discs may be the only means of arriving at a positive conclusion.

3. That there is no organic disease of the heart, arteries, or kidneys—conditions which predispose to vascular rupture, embolism and thrombosis, and hence to organic paralysis.

B. *Positive Evidence.*

1. The fact that the patient is a female, generally young; and that she presents symptoms and signs of hysteria—such as emotional tendencies, globus hystericus, tenderness on pressure over the ovarian region, a history of recent or past hysterical fits, &c.

The appearance of the face in cases of hysterical paralysis is often characteristic, but difficult to describe. According to Todd the *facies hysterica* is as follows: "An expression characterised by a remarkable depth and prominent fulness, with more or less thickness of the upper lip, and of a peculiar drooping of the upper eyelid."

2. *Irregular Distribution and Character of the Paralysis.*

There may be nothing peculiar in the distribution of the paralysis in hysteria. In many cases, however, it is irregular; the leg affected more than the arm in hemiplegic cases; one arm and the opposite leg; part of a limb only. In hysterical hemiplegia the face and tongue are very rarely involved. In hysterical hemiplegia, too, the leg is dragged as a dead weight, and is not circumducted. Todd laid great stress upon this point, as the following quotation shows:—"But I would particularly call your attention to the peculiar character of the movement of the paralysed leg when the patient walks, which, in my opinion, is characteristic of the hysterical affection. If you look at a person labouring under ordinary hemiplegia from some organic lesion of the brain, you will perceive that, in walking he uses a particular gait to bring forward the paralysed leg; he first throws the trunk to the opposite side, and rests its entire weight on the sound limb; and then, by an action of circumduction, he throws forward the paralysed leg, making the foot describe an arc of a circle. Our patient, however, does not walk in this way; she drags the paralysed limb

after her, as if it were a piece of inanimate matter, and uses no action of circumduction, nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks. This I believe to be characteristic of the hysterical form of paralysis."[1]

The *onset* in hysterical cases is often quite sudden, the paralysis appearing abruptly after an hysterical fit or some emotional disturbance. This fact is not of very great value, for organic paralysis also very commonly sets in suddenly, and the exciting cause may be a fright, or other emotional disturbance.

The *course* in hysterical paralysis is very variable. Usually the duration is short, the case terminating suddenly in complete recovery. In other cases the paralysis lasts for years.

Sudden improvements, then, and sudden relapses are characteristic of hysterical paralysis. In some cases of cerebro-spinal sclerosis (sclérose en plaques) the same fact—sudden improvement and sudden relapse—is observed. I have known such a case, for this reason, thought to be hysterical.

In cases of hysterical paralysis associated with contracture, the paralysis and rigidity appear together quite suddenly, differing in this respect from organic contracture, i.e. the late rigidity of Todd. The contracture, too, in hysterical cases, is apt to involve the leg as much as, or more than, the arm. In organic cases (hemiplegia with contracture) the rigidity is often limited to the arm, and never involves the leg alone. In hysterical cases where the contracture involves the leg, the toes are pointed, and the foot somewhat inverted.

According to Charcot, chloroform relaxes hysterical, but not organic contracture. This statement obviously only applies to chronic cases in which the organic contracture is associated with secondary (cirrhotic) changes in the affected muscles.

3. *The Condition of the Sensory Nerve Apparatus.*

At the commencement of organic cases there is generally more or less disturbance of sensibility (anæsthesia), but in the great majority of cases the anæsthesia soon passes off—much more rapidly than the paralysis. In organic cases there is seldom complete hemi-anæsthesia. (An exception to this occurs in those cases in which a lesion involves the posterior part of the *internal capsule* or adjacent parts. In those cases

[1] 'Clinical Lectures on Paralysis,' p. 20.

the motor paralysis is often slight, for the simple reason that, the lesion is not sufficiently extensive to destroy the motor fibres too.)

In hysterical cases, on the contrary, there is usually great disturbance of sensibility. In some, profound anæsthesia, often strictly limited to, but involving all the parts, including the special senses, on one side of the body. In others, sensibility to pain is completely abolished, but sensibility to touch remains. Again, there may be hyperæsthesia, the slightest touch producing complaints of pain and suffering.

4. *The condition of the bladder and urine.*—In hysterical paralysis, retention requiring the use of the catheter is often a prominent symptom. It usually continues so long as the instrument is passed by the House Surgeon, but often yields when his place is taken by a female nurse. The *urine* in hysterical cases, especially after a "fit" or other emotional manifestation, is pale, limpid, and very abundant. In some cases there is ischuria.

5. *The condition of the uterus and ovaries.*—In hysterical cases there is frequently some disorder of the uterine and ovarian functions. Tenderness on pressure over the ovaries is sometimes observed. Slight pressure, according to Professor Charcot, induces an hysterical fit, while firm pressure arrests it. These effects of pressure are not, however, as a rule, seen in the hysterical cases met with in this country.

6. *The previous history.*—A history of previous hysteria, and especially of previous attacks of paralysis which have been rapidly and completely recovered from.

Epileptic Paralysis.

Epileptiform paralysis is a functional paralysis. It occurs in those cases of epilepsy which depend upon the presence of a "coarse" (cortical) lesion, and is rarely, if ever, seen after the convulsions of the so-called "genuine" or "idiopathic" disease. The paralysis is limited to the muscles which were convulsed (or most convulsed); and, since "coarse" cortical lesions are generally unilateral, or limited in extent, the resulting paralysis and convulsions are unilateral, and often limited to one limb, to one group of muscles, or even to one muscle.

The paralysis is supposed to be due to the exhaustion of grey matter (motor nerve cells of the cortex) which follows the excessive and violent, or, as Dr. Hughlings-Jackson expressively puts it, "brutal," discharge which causes the convulsion. Granting, as I do, that this explanation is probably true, it is a remarkable fact that paralysis does not occur after the convulsions of the *idiopathic* disease, in which the discharge, as measured by the severity of the spasms, is very violent.[1]

Epileptic paralysis is quite temporary, passing off after a few hours or days at the most.

Organic paralysis may be associated with convulsions. In some cases of cerebral hæmorrhage the attack is ushered in by convulsions and followed by paralysis. Paralysis and convulsions are also observed in some cases of embolism. It is, therefore, a most important question to decide whether in any given case of convulsions and paralysis, the loss of motor power is of the temporary (functional) kind, or whether it is due to an organic lesion, i.e. permanent. If we can decide in favour of the functional variety, we are able to give great mental comfort to the patient and to his friends; for paralysis is an objective symptom, which is, naturally, regarded by the laity as most ominous. In cases of epileptic paralysis, motor power is, as I have already stated, quickly regained, and under appropriate treatment (iodide of potassium in full doses) the general condition of the patient often improves in a remarkable degree. Great credit and reputation are often, therefore, to be made by the satisfactory recognition and successful treatment of such cases.

The points on which a diagnosis of epileptiform paralysis is to be based are the following:—

1. The fact that the paralysis *followed* a fit (epileptiform convulsion), presenting the characteristic features of the convulsions which result from the presence of a "coarse" cortical lesion:

   (*a*) The spasms have a local commencement, and are often limited to one set of muscles, or to the muscles

[1] I have attempted to explain this fact by supposing that there is a difference in the process of discharge in the two cases. See 'Edinburgh Medical Journal,' March, 1881, page 831.

on one side of the body. In some cases the spasms after commencing locally become general (bilateral).

(*b*) The attack is not attended with loss of consciousness, or rather that, so long as the spasms are local, there is no loss of consciousness; for, when the spasm becomes general, consciousness is usually lost, just as in a typical epileptic fit.

2. The fact that the paralysed muscles are those which were most and first convulsed. This is all-important, for it shows that the conducting fibres from the centres to the muscles are sound, and that the lesion is situated in the discharging centre itself. It is not, of course, always possible to say from personal observation which muscles were convulsed. We may therefore have to rely upon the statement of the patient or his friends for information, and such information cannot unfortunately be always implicitly relied upon.

We have now excluded hæmorrhagic apoplexy. (In a case of cerebral hæmorrhage sufficiently severe to be attended with convulsions, consciousness is profoundly affected for some time. The spasms do not recur, or if they do recur, they do not affect the paralysed muscles. There are the other features of a severe apoplectic attack.) And we have to determine whether the paralysis depends upon embolism or thrombosis, or whether it is epileptic, i.e. functional; for in embolism and thrombosis convulsions, unattended by loss of consciousness, may be present in addition to the paralysis, the anæmia or subsequent softening being the common cause of both conditions.

The differential diagnosis of a "coarse" cortical lesion, i.e. of such a lesion as we know by experience is the cause of epileptiform paralysis, on the one hand, and of an embolism or thrombosis on the other, is to be decided by attention to the following additional points:

1. *The presence of the symptoms and signs of a "coarse" cortical lesion.*

(*a*) Headache, usually severe, frequently localised, often paroxysmal, and in such cases sometimes associated with tenderness on skull percussion.

(*b*) Vomiting of a purposeless (cerebral) character.

(*c*) Double optic neuritis or optic atrophy. This symptom is of the greatest importance, for it is present in the majority of cases of "coarse" lesion (tumour), but does not occur in embolism or thrombosis.[1]

2. *The history and mode of commencement of the attack.*—In embolism the convulsions and paralysis occur abruptly, and there may have been no previous cerebral symptoms. In thrombosis there is sometimes a history of vertigo, headache, and evidence of slight sensory and motor disturbance.

In the case of a "coarse" lesion (tumour) there is almost invariably a history of previous cerebral symptoms (i.e. the occurrence of headache, &c., before the attack of convulsions and paralysis).

3. *The associated conditions.*—In the case of a "*coarse*" *lesion* there are often associated lesions of a similar nature (syphilitic, tubercular, cancerous, &c., as the case may be). In embolism there is the presence of cardial or arterial (aortic) disease. The occurrence of the attack during pregnancy, &c. *In thrombosis* the arterial system is diseased, the superficial arteries are usually atheromatous, the patient is, as a rule, past middle age. (In syphilitic cases there may be no evident disease of the superficial vessels, and the patient may be young.)

4. *The effects of treatment.* Epileptiform paralysis speedily disappears; the paralysis due to embolism and thrombosis

[1] In cases of limited meningitis of the convexity, we have a "coarse" lesion, which is usually unattended with optic neuritis. There is fortunately not much difficulty in deciding between such a lesion on the one hand, and embolism or thrombosis on the other. Localised meningitis is either traumatic or tubercular. In the former case the history, in combination with the symptoms, is sufficient to decide the point. In tubercular cases the question is decided by:

1. *The character of the symptoms.*
   (*a*) Headache, usually a prominent symptom in meningitis; often absent in embolism and (though less frequently so) in thrombosis.
   (*b*) Vomiting. Much more frequent in meningitis.
   (*c*) The condition of the temperature. More usually elevated in meningitis than in embolism.
2. *The mode of commencement of the attack.* See text, page 40 (2).
3. *The associated conditions.* See text, page 40 (3).

Dr. Stephen Mackenzie has reported two cases of embolism in which there was double optic neuritis; but this is so rare that the general statement in the text may be safely relied upon.

remains. The general symptoms, too, in many cases of cerebral tumour (syphilitic cases) improve rapidly under appropriate treatment (full doses of iodide of potassium).

Paralysis depending upon Idea.

Under the term "Paralysis depending on idea," Dr. Russell Reynolds has described certain cases of paralysis depending upon imagination. In these cases there is no intention to deceive; the patients really believe that they are the victims of serious organic disease. Cases of this sort are quite distinct from ordinary cases of hysterical paralysis and from ordinary cases of hypochondriasis. The patients are for the most part of a highly, and often very active, mental nervous temperament; their general health is usually below par, but they do not, so far as my limited experience goes, exhibit the usual symptoms of hysteria. The fixed belief that they are paralysed; and the concentrated attention which the affected parts receive, together with the effect, which is produced on the mind by such a state of matters, induces functional disturbances often of a striking character. Startings, twitchings, and fibrillary tremors, occur in the limbs and other parts of the body. Aching muscular (myalgic) pains are common. The heart is easily excited; the patient suffers from attacks of palpitation. Exertion or mental excitement is followed by a feeling of exhaustion and fatigue. The patient becomes impressed with the idea that he is unable to do anything; that he is paralysed, &c. There is often sleeplessness and restlessness. The patient wakes up towards the early morning hours, and rises unrefreshed. The stomach may be deranged; constipation is common. On physical examination the reflexes—superficial and deep—are generally found to be exaggerated, the affected parts (muscles supposed to be paralysed) are usually soft and flabby; in chronic cases there may be considerable atrophy, but it is not limited to special muscles or groups of muscles; a point of importance, for, in some cases which have come under my notice, the diagnosis lay between this condition and progressive muscular atrophy. *Sensibility* is not affected. The *bladder* is healthy; the bowels, as already mentioned, are usually constipated.

The loss of motor power is never complete, and often presents anomalous characters; for example, the patient, who can neither stand nor walk, will move the legs in any direction when in bed, &c.

This condition often lasts for a long time, it may be years; and is sometimes most difficult to cure.

The diagnosis is to be made by attention to the following points:—

1. *The condition of the paralysed parts.*—There is no positive evidence of organic paralysis.

2. *The mental condition and temperament of the patient.*—The fact that the patient's mind is concentrated on his condition; that he is always thinking of his symptoms; examining his limbs, &c.; and that he is thoroughly impressed with the idea that he is the subject of serious organic disease.

3. *The condition of the reflexes.*—The increased reflex excitability, and the fact that it is general, and is not confined to the paralysed parts, is, so far as my limited observation enables me to judge, a diagnostic point of some value.

4. The fact that the startings, jerkings, and fibrillary twitchings are not limited to special muscles, and that the muscles in which they occur do not present any special atrophy.

5. The fact that there is no affection of the bladder or rectum.

6. *The history and progress of the case.*—The fact that the case remains stationary for years, and especially that no marked alterations in the condition of the paralysed parts appear.

In some cases there is a family history of genuine disease of a similar kind. This fact, of course, cuts both ways; for, on the one hand, we know that nerve affections are frequently hereditary, on the other, it must always be remembered that persons of a susceptible nervous temperament, who are brought in contact with disease, are very apt to imagine that they are themselves the subjects of it.

7. *The effects of treatment.*—A favourable opinion, confidently expressed, and appropriate treatment, sometimes effect rapid and remarkable improvement.

Malarial Paralysis.

Temporary paralysis sometimes occurs as the result of malarial infection. The pathology of the condition is unknown. Cases of this sort are rare, even in malarial districts, and no case has come under my personal observation. The characteristic feature of the paralysis is that it is intermittent, and that it occurs at regular intervals, just as the ordinary febrile paroxysms (quotidian, tertian, &c.) do.

The diagnosis is founded upon:

1. The absence of the signs and symptoms of organic disease.
2. The intermittent character of the paralysis.
3. The fact that the paralysis is cured by antimalarial remedies (quinine).

Reflex Paralysis.

Functional paralysis occasionally results from peripheral irritation acting reflexly. The source of the irritation is usually in the bladder or urethra.

Brown-Séquard supposes that the immediate cause of the paralysis is anæmia of the cord, and that this is due to spasmodic contraction of the vessels of the cord produced reflexly. In many cases of so-called reflex (urinary) paralysis there are organic changes in the cord of an inflammatory character. In some of these cases, probably the majority, the inflammatory changes result from direct extension to the cord of similar conditions in the distant (peripheral) parts. The extension may take place along the nerves (neuritis ascendens) or through the blood-vessels.

In some cases organic lesions have been found in the cord, when the intermediate parts have appeared to be healthy, and it is supposed by Benedict and others that, organic spinal lesions can actually be induced reflexly, independently of direct extension.

The cases in which there is an organic lesion of the cord cannot correctly be termed *reflex*, if we understand, as I do by that term, *a functional paralysis due to peripheral irritation acting reflexly.*

True reflex paralysis is undoubtedly rare, but that it does

occasionally occur in man seems beyond dispute; and that it can be induced in the lower animals seems proved by the remarkable chloroform experiments which Brown-Séquard has lately published. The diagnosis of reflex paralysis is always hazardous, and should only be made when:

1*stly*. There is no evidence of organic disease.

2*ndly*. There is a manifest source of peripheral irritation.

3*rdly*, and *chiefly*. The removal of that irritation is followed by the disappearance of the paralysis.

Temporary paralysis due to Vasomotor changes in the cord are sometimes met with, and are clearly functional in character.

Step No. 3.—*The paralysis is organic, what is the position and extent of the lesion* (anatomical diagnosis), *and what is its character* (pathological diagnosis)?

In seeking to determine the position and extent of the lesion, we must, in the first place, decide whether the paralysis is peripheral, spinal, or cerebral (including under that term pontine and medullary paralyses), and having determined this point we must, in the second place, endeavour to ascertain the particular part of the nerve-trunk, spinal cord, or brain, respectively, which is affected.

The points to which attention is to be directed in making the anatomical diagnosis are:

1. The exact distribution of the paralysis.
2. The condition of the paralysed muscles, especially as regards "rapid atrophy" and the "reaction of degeneration."
3. The condition of the reflexes, superficial and deep.
4. The condition of sensibility.
5. The associated nerve symptoms—more particularly derangement of parts indicating lesions of any particular part of the nervous system (localising spinal and cerebral symptoms).
6. The presence of any obvious cause, such, for example, as a traumatic injury of the skull, spine, or nerve-trunk.

ON THE CAUSE OF THE MOVEMENTS OF ORDINARY RESPIRATION. ARE THESE MOVEMENTS REFLEX?

BY AUSTIN FLINT, JUN., M.D.

Professor of Physiology in the Bellevue Hospital Medical College, New York, &c.

THE movements of ordinary respiration, which begin at birth and continue uninterruptedly throughout the life of man and of all warm-blooded animals under normal conditions, have an exciting cause. This cause, whatever it may be, does not operate during the period of intra-uterine existence; and it is well known to physiologists that the part which the lungs play after birth in the introduction of oxygen is performed in the fœtus by the placenta. If the umbilical vessels be compressed in a living animal, the fœtus will soon begin to make respiratory efforts; and this even at an early stage of development. This interesting experiment is by no means new, and it is one that I have often repeated. Another very instructive observation, illustrating the same principle in adult animals, was made in 1664 by the celebrated Robert Hook. In this experiment, it was ascertained that, if air be efficiently supplied to the lungs of a living animal by artificial respiration, as by fixing a bellows in the trachea and regularly inflating the lungs at short intervals with fresh air, respiratory efforts will soon cease, and the animal will remain perfectly quiet so long as the artificial supply of air is properly maintained.

In the first of the experiments above-mentioned, movements are apparently excited because the respiratory function of the placenta is interrupted; in the second, the normal respiratory movements are arrested, as it seems, because the exciting cause of these movements is temporarily removed. The explanation of the phenomena observed in both experi-

ments involves the assumption of the existence of some exciting cause or of a demand on the part of the system which secures, in the adult animal, the regular and periodic introduction of fresh air into the lungs. In normal, tranquil respiration, this cause operates independently of sensation. When there is any deficiency in the supply of fresh air, one evinces a sense of respiratory difficulty, or it may be exaggerated to the point of a feeling of impending suffocation. This sense, both normal and exaggerated, might properly be described as the sense of want of air, or, as it is called by the French, *besoin de respirer*. Many years ago, about 1809, Legallois showed that animals instantly ceased to breathe when the medulla oblongata was destroyed. In 1823 and 1827 Flourens clearly defined a certain portion of the medulla oblongata, near the origin of the pneumogastric nerves, as the respiratory nervous centre. Their experiments and those made subsequently by other physiologists demonstrated conclusively that a nerve-centre situated in the medulla oblongata is the only part capable of appreciating the sense of want of air. When this part is destroyed, respiratory movements instantly cease, for the simple reason that the nerve-centre, which alone is capable of receiving the impression due to want of air, is destroyed. About 1833 the attention of physiologists was directed by Marshall Hall to what are now known as reflex phenomena. He regarded the respiratory movements as reflex, depending upon an impression conveyed to the medulla oblongata through the pneumogastric nerves, received by the medulla oblongata and reflected back through certain motor nerves to the muscles of inspiration.

This brief historical sketch, in which only the most important of the many experiments made on the subject under consideration have been mentioned, serves to show the state of physiological knowledge up to a time a few years later than the observations of Marshall Hall.

In following out the question of the cause of the respiratory movements, it is necessary to appreciate as exactly as possible the mechanism of the appropriation of oxygen by the system.

With each inspiratory act, about twenty cubic inches of fresh air are taken into the respiratory organs to replace about

the same quantity of vitiated air expelled in expiration. The air thus introduced is lighter than the air contained in the deeper part of the lungs, the latter being charged with carbonic acid. It is evident, however, that little if any part of the twenty cubic inches of fresh air can immediately reach the air-cells, in which the interchange of gases between the air and the blood actually takes place, for the lungs ordinarily contain about two hundred cubic inches; but, in accordance with the law of diffusion of gases, there is a constant progression of the air, laden with carbonic acid, from the deeper parts of the lungs toward the trachea, and an equally constant penetration of the fresh air toward the air-cells. In this way, although the fresh air is introduced and the vitiated air is expelled intermittently about eighteen times per minute, the actual contents of the air-cells probably present a pretty constant composition as regards oxygen and carbonic acid; the supply of oxygen being maintained by the repeated inhalation of fresh air, and the blood, in its passage through the pulmonary capillaries, constantly giving off carbonic acid. It is only when the supply of oxygen is deficient that one is actually conscious of a sense of want of air.

There is a corresponding regularity in the current of blood through the capillary vessels in the walls of the air-cells. The heart contracts intermittently about seventy times per minute; but the successive charges of blood that are sent by the right ventricle into the pulmonary artery are received by vessels of great elasticity, which, as it were, absorb the intermittent force of the heart, so that the current which passes through the capillary vessels of the lungs is constant and of uniform rapidity. The venous blood thus passing through the lungs is constantly exhaling carbonic acid into the air-cells and receiving oxygen. The oxygen thus taken up by the blood immediately forms a union with the colouring matter of the blood-corpuscles, the blood becomes oxygenated or arterialized, and is distributed to the system through the branches of the aorta. As the blood passes into the capillaries of the general system, the same cause which secures a constant and uniform current of blood through the pulmonary capillaries, viz. the elasticity of the arteries, produces a constant

and uniform flow of blood, which gives up its oxygen to the tissues and passes into the veins, laden with carbonic acid. The lungs simply serve to present oxygen to the blood; and the blood is the vehicle by which oxygen is carried to the tissues; true respiration, however, consists in the appropriation of oxygen by the tissues, and is constantly going on in every highly organised part in the economy.

The theory of Marshall Hall, that respiratory movements are excited by the accumulation of carbonic acid in the lungs, the impression thus produced being conveyed to the medulla oblongata by the pneumogastric nerves, is disproved by the fact that these movements continue, although modified, after section of the pneumogastrics in the neck; and, as early as 1839, John Reid suggested that the sense of want of air was due in a measure to the circulation of venous blood in the medulla oblongata itself. In 1841, Volkmann made a number of experiments, from which he concluded that the respiratory movements were reflex in their character, but were due to the stimulation by carbonic acid of afferent nerves in every part of the body. Without entertaining any doubt with regard to the reflex character of the respiratory movements, I made, in 1861, a series of experiments, in which I endeavoured to show that the sense of want of air was due to want of oxygen in the general system, and not to the stimulation of afferent nerves by carbonic acid. In these experiments, I first showed that in living animals, after the respiratory movements had been arrested by artificial respiration, respiratory efforts began (when artificial respiration was interrupted) only when the blood became dark in the arteries. Recognizing the fact that oxygen can reach the tissues only through the blood, I drained the animal of blood, still keeping the lungs supplied with air, and always succeeded in this way in exciting respiratory movements. The views which I then entertained were still further supported by experiments made by Pflüger, in 1868, who excited respiratory movements in animals by insufflating the lungs with an irrespirable gas, such as pure nitrogen.

Having often repeated my experiments since 1861, frequently as class demonstrations, it occurred to my mind in 1877 that possibly the respiratory movements might be due to some

direct change in the conditions of the medulla oblongata, and I began to entertain doubts with regard to their reflex character. I then undertook a series of experiments which led me to the conclusion that the sense of want of air is due to the want of oxygen in the medulla oblongata itself. The general results of these experiments, the details of which have been published elsewhere, were as follows:—

In a dog brought fully under the influence of ether, artificial respiration was established by means of a bellows fixed in the trachea so completely that all respiratory efforts on the part of the animal ceased. The innominate artery and the left subclavian artery were then exposed so that the vessels could be constricted at will. The irritability of the medulla was tested by interrupting artificial respiration, which was followed by respiratory efforts. Then, artificial respiration having been resumed so that the animal remained perfectly quiet, the great vessels given off from the arch of the aorta were constricted, the artificial respiration being continued. This was invariably followed by violent respiratory efforts, which began in a little more than two minutes after constriction of the vessels and continued until the vessels were freed, when the efforts ceased. No such phenomena followed constriction of the aorta below the arch, which, of course, shut off the blood from all parts of the body except the head and anterior extremities. The experiments of which this is an example were frequently repeated, always with the same results. Of course, if it be assumed that the medulla oblongata is the sole respiratory nerve-centre, it is reasonable to suppose that the occlusion of the vessels given off from the arch of the aorta, which cuts off the supply of oxygenated blood from the medulla, gives rise to a sense of want of air by reason of some change in the conditions of the medulla, which conditions are again changed so soon as blood is allowed to flow again through these vessels.

Reasoning from the facts developed by my own experiments, taken in connection with what is well known and established with regard to the action of the medulla as a respiratory nerve-centre, the following seems to be a satisfactory explanation of the mechanism of the ordinary respiratory acts:—

The left ventricle sends arterial blood received from the lungs to all parts of the system, including the medulla oblongata. The elasticity of the aorta and of its branches gradually extinguishes or absorbs the intermittent force of the heart, so that the blood flows in a steady and continuous stream through the capillaries of the medulla. But, as the tendency of the air in the pulmonary cells is to progressively increase in its proportion of carbonic acid and to diminish in its proportion of oxygen between two respiratory acts, the tendency of the blood coming from the lungs and sent by the left ventricle to the medulla oblongata is to become progressively poorer in oxygen. After about four revolutions of the heart, assuming that the proportion of the beats of the heart to the respiratory acts is four to one, the quantity of oxygen supplied to the medulla oblongata has become so far diminished that there occurs an unconscious sense of want of air, and this excites a new inspiratory act. So it is, in all probability, that the normal rhythmical acts of inspiration are periodically excited; and anything, like violent muscular exercise, that increases the activity of the consumption of oxygen, of necessity increases the number of respirations per minute.

When there occurs any serious interference with the passage of fresh air to the air-cells or an obstruction to the flow of arterial blood to the medulla oblongata, as in certain pulmonary and cardiac diseases, the unconscious sense of want of air is exaggerated until one becomes conscious of pulmonary oppression or impending suffocation. This is simply an exaltation and extension of the normal respiratory sense so that it reaches the true sensory centres, causing a voluntary increase in the number and extent of the respiratory acts. The sense of suffocation, indeed, differs from the normal respiratory sense merely in degree and in the fact that the former operates on the centres of ordinary sensation, while the latter is confined to the medulla oblongata.

When respiration has been so long obstructed that respiratory efforts cease, the medulla rapidly loses its capacity to appreciate the sense of want of air; still, under these circumstances, if the heart continue to beat, artificial respiration, if persisted in so as to restore the supply of arterial blood to the

medulla, will often restore the sensibility of the respiratory nerve-centre, so that finally the respiratory movements will become re-established. Physiologists who are in the habit of administering anæsthetics to animals have frequent occasion to note this fact. A dog, for example, becomes so overpowered by the anæsthetic that the sensibility of the medulla is for the time destroyed and respiration is arrested; the heart, however, continues to act, though its contractions are feeble; but artificial respiration, if kept up efficiently and persistently, will maintain the action of the heart, the respiratory sensibility of the medulla gradually returns, and after a time the respiratory movements are re-established.

Narcotics also may affect the respiratory sensibility of the medulla so that the frequency of the respiratory acts is diminished and they may be arrested; and in such instances it is sometimes possible to revive the respiratory function by artificial insufflation of the lungs. In most cases of suspended respiratory action from any temporary cause, although galvanism, sudden and active stimulation of the surface, &c., may aid in restoration, the main reliance should be upon persistent and efficient artificial respiration.

Are the Normal Respiratory Movements either entirely or in part Reflex, in the Sense in which the term Reflex is ordinarily understood by Physiologists?

I shall leave out of this question various modifications of the respiratory acts, such as coughing, sneezing, &c., and the influence of certain unusual impressions made upon the general surface, as by a cold douche, and restrict the discussion to the phenomena of ordinary respiration. Experiments have shown that the unconscious and automatic movements of respiration in an animal can be abolished by artificially supplying the lungs with fresh air, which has the effect of securing to the capillaries of the medulla oblongata as well as of other parts a sufficient quantity of oxygenated blood. This supply of oxygen through the blood removes the exciting cause of the respiratory movements by abolishing for the time being the sense of want of air. When, however, there is a deficiency in the supply of oxygen to the medulla, this deficiency produces

a sense of want of air, and respiratory efforts follow. The quantity of blood sent to various parts of the body is subject to frequent variations through the influence of the vaso-motor nerves upon the muscular coat of the arteries; in other words, these nerves may modify and regulate local circulation independently of the action of the heart; but the circulation in the medulla oblongata seems to be the physiological gauge of the requirements of the general system for oxygen; and it is by virtue of this property that the medulla is enabled to act as the respiratory nerve-centre. Every vascular part of the body undoubtedly requires oxygen, a proper supply of which is necessary to its nutrition and to the performance of its functions. Normal variations in nutritive or functional conditions may and do involve in certain parts great modifications in the supply of blood, which modifications are regulated by vaso-motor nerves; but these variations in the local circulations do not usually produce impressions capable of modifying the respiratory acts, unless they be attended with an actual increase in the quantity of oxygen consumed in the organism. In case the consumption of oxygen be increased from any cause, the quantity supplied by the lungs must also be increased in order to keep the proportion of oxygen in the arterial blood at the proper standard. The respiratory nerve-centre seems to take cognisance promptly of any deficiency in the supply of oxygen to its substance, and it measures by this deficiency the respiratory demands of the system. With a knowledge of the results of recent experiments, one can hardly imagine that the different parts of the organism, possessing, as they do, such a variety of functions and properties, and subject to such modifications in the supply of blood which they receive, can all be endowed with a common sense capable of being conveyed to the medulla oblongata and there appreciated as the sense of want of air. With the variations to which the circulation in special parts is subjected, it would seem that a confusion of impressions must necessarily result, if these impressions originated in the general system. It is only in the pulmonary structure, indeed, that any such impression could by any possibility arise; but this is not the fact, for the following reasons: Respiratory movements occur when the nervous connection between the

lungs and the medulla has been severed. When the supply of oxygenated blood to the medulla has been cut off, respiratory efforts occur, even though the lungs be fully supplied with air and no deficiency of oxygen or excess of carbonic acid can exist in the air-cells.

Taking into consideration all the facts bearing upon the question, I can come to but one conclusion with regard to the character of the movements of normal, tranquil respiration.

When perfectly normal, and not modified by any unusual physiological conditions, the regular acts of the tranquil respiration are not reflex, at least in the sense in which the term reflex is ordinarily understood by physiologists. As I have said before, there is a constant and uniform current of arterial blood to the medulla oblongata, and there is a progressive diminution in the proportion of oxygen in the blood, between the respiratory acts. After a certain time, the supply of oxygen to the medulla becomes so far reduced that it gives rise to an unconscious sense of want of air, which excites an act of inspiration. This repeats itself regularly from eighteen to twenty times in a minute. Under these conditions, regular respiratory movements are excited by a stimulus generated in the medulla oblongata itself, and depending solely upon the state of the circulation in this part.

Normal Modifications of the Respiratory Movements by Reflex Action.

Although regular movements of respiration may go on without any action that can strictly be called reflex, reasoning with regard to respiration from the phenomena observed in connection with other important functions, one would naturally expect to find the process by which oxygen is introduced into the body subject to modifying and regulating influences operating through the nervous system. The action of the heart is undoubtedly automatic, but the force and frequency of the contractions of this organ are certainly controlled more or less, under physiological conditions, through the nervous system. The connection between aëration of the blood and the circulation is certainly very close; physiological conditions must frequently occur which demand changes in the rapidity

and extent of respiratory acts without involving voluntary effort or even consciousness; and many of these demand the intervention of nervous action aside from the mere development of the sense of want of air in the medulla oblongata. Admitting the truth of my propositions, as regards perfectly tranquil respiration, it is well known that important physiological reflex influences upon the acts of respiration operate through the pneumogastric nerves.

A relatively strong galvanic current applied to the pneumogastric nerves in the neck, or to certain branches of the pneumogastrics (the superior and the inferior laryngeals), will instantly arrest respiration. This action is reflex, as is shown by the fact that galvanisation of the central ends only, of the divided nerves influences respiration. The fact just stated is marked and constant; at the same time, it has been noted that galvanisation of certain other sensory nerves will arrest respiration, although this result is not invariable. When the respiratory movements are completely arrested by galvanisation of the pneumogastrics, it is always the same for the general movements of the animal. On the other hand, "a feeble excitation accelerates the respiration; a more powerful excitation retards it; a very powerful excitation arrests it. These words 'feeble' and 'powerful' having, it is understood, only a relative sense for any one animal, and under certain conditions: what is feeble for one would be powerful for another," &c. (Bert.)

As far as these experimental facts can be applied to the physiology of ordinary respiration, it seems that the nerves, the action of which is brought into play under physiological conditions, must be mainly, if not exclusively, the pneumogastrics. These nerves have their origin at the medulla oblongata; they are distributed to the entire respiratory apparatus, from the larynx to the deepest parts of the lungs, but not to the respiratory muscles, except the intrinsic muscles of the larynx; and they are capable, by reflex action, of exerting a very marked influence over the respiratory movements.

It is important, in this connection, to note another experimental fact with regard to the pneumogastrics. When both of these nerves are divided in the neck, the excitation directly

produced by their section momentarily accelerates the respiratory movements. In animals in which the walls of the larynx are sufficiently rigid to enable the acts of inspiration to be carried on without any serious obstruction, the following phenomena are observed:—For a few seconds, rapidity of the respiratory movements may be increased, but so soon as tranquillity is restored, the number of respirations per minute is very much diminished, falling from sixteen or eighteen to four or five, and the inspiratory acts become unusually profound, and are attended with excessive dilatation of the thorax.

The respiratory phenomena following galvanisation and section of the pneumogastrics in the neck, have never been satisfactorily explained; and certainly such an explanation would throw some light upon the reflex action of these nerves in normal respiration. If, however, my view of the action of the medulla oblongata in tranquil respiration be accepted, a very interesting proposition made by Rosenbach, in 1878, renders it possible to present a theory which, while it may not be entirely satisfactory, is interesting and suggestive, and may be sustained by future experimental observations. Rosenbach advances the idea that the pneumogastrics are the vaso-motor nerves of the medulla oblongata, and that they contain fibres which contract and fibres which dilate the blood-vessels. This is presented merely as an hypothesis, which he "hopes later to be able to establish by experiments."

If it be assumed, for sake of argument, that the pneumogastrics actually contain fibres which regulate the supply of blood to the medulla oblongata, it would be easy to understand how such fibres could so influence the supply of oxygen to the respiratory nerve-centre as to secure a succession of respiratory acts such as would be demanded by the system under different physiological conditions; still, the seat and the exact nature of the impressions giving rise to reflex changes in the calibre of the vessels of the medulla would be a matter of speculation and conjecture.

Applying the suggestion made by Rosenbach to experiments upon the pneumogastrics, the phenomena observed could be explained as follows:—

Relatively feeble galvanisation of the pneumogastrics ac-

celerates respiration by contracting the vessels of supply of the medulla, and thus diminishing the supply of oxygen.

Powerful galvanisation of the pneumogastrics arrests all movements in animals, including the respiratory movements.

When the action of the medulla is removed from the influence of the pneumogastric nerves, as it is after the division of both nerves in the neck, air is taken into the lungs when the deficiency of oxygen in the medulla has reached the point at which the sense of want of air necessarily generates the stimulus sent to the inspiratory muscles. This action is in no sense reflex, and it depends entirely upon the development *de novo* of a stimulation or an irritation in the medulla itself. Under these conditions, the acts of inspiration are abnormally infrequent, and they become excessively prolonged and profound. It is probable that death occurs in a few days after this operation, not alone from abnormal respiratory action, but from a suspension of other important functions which the pneumogastrics have to perform. It is possible, also, that some of the phenomena which are observed in narcotic poisoning—notably a great diminution in the frequency of the respiratory movements—may be due in part to an interference with the respiratory functions of the pneumogastrics.

As far as muscular action in tranquil respiration is concerned, I have thought it necessary to consider only the acts of inspiration; for expiration is produced mainly by the passive contraction of the capacity of the thorax, and by the resiliency of the elastic pulmonary parenchyma succeeding the action of the muscles which enlarge the chest and inflate the lungs.

The results of my experiments, observations, and reflections upon the cause and nature of the movements of normal respiration may be embodied in the following propositions:—

1. The respiratory sense, *besoin de respirer*, sense of want of air, or the stimulus which gives rise to respiratory efforts, is due purely and simply to a deficiency of oxygen in the medulla oblongata, which is the sole respiratory nerve-centre.

2. In perfectly tranquil, uniform, and undisturbed respiration, the regular and successive inspiratory acts may go on in

obedience to successive stimuli, originating *de novo* in the medulla oblongata. Such respiratory movements are not reflex.

3. The frequency and the extent of the normal inspiratory movements are regulated and accommodated to the physiological requirements of the system by reflex action operating through the pneumogastric nerves.

ON SOME POINTS IN THE DIAGNOSIS AND TREATMENT OF BRAIN DISEASE.

BY JULIUS ALTHAUS, M.D., M.R.C.P.

Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park.

(Concluded from Vol. III. p. 308.)

WHILE therefore our knowledge of the clinical features of thalamus opticus lesions is as yet very fragmentary, we possess a large body of evidence with regard to the distinctive characters of disease in the neighbouring ganglionic mass of the *corpus striatum*, the several portions of which were first clinically differentiated by the observations of Tuerck, Meynert and Charcot, as they had long before been anatomically unravelled by Burdach in his great work on the structure of the brain. The prominence given, and the great interest attaching to the affections of the different portions of the corpus striatum, which are more frequent and practically important than any other localised brain-lesions, have caused the terms of lenticular and caudate nucleus, and external and internal capsule,[1] to become household words in neurological literature.

The term "corpus striatum" is now used in a somewhat different sense by various writers. Most pathologists comprehend by it (*a*) the grey caudate nucleus, Schwanzkern, or intraventricular portion of the ganglion, which comes in sight on laying bare the lateral ventricle; (*b*) the lenticular nucleus, Linsenkern, or extraventricular portion, which is imbedded in the cerebral substance, and therefore not visible from the ven-

[1] Singularly enough, the terms "external and internal capsule" are nowhere to be found in the last edition of 'Quain's Anatomy,' by Sharpey, Allen Thomson and Schäfer. (1878.)

tricle; and (*c*) the white internal capsule, which is situated between the two nuclei, and forms the connecting link between the crus cerebri on the one hand, and the corona radiata and the cortex, of the brain on the other hand. Prof. Dalton,[1] of New York, and others have recently restricted the term corpus striatum to the caudate nucleus or intraventricular portion of the ganglion; while in the following remarks, for the sake of clinical convenience, both nuclei, the internal and external capsule, and the claustrum, will be comprehended under the name of corpus striatum. The claustrum (Vormauer, avant-mur) is that thin layer of grey matter which separates the lenticular nucleus from the convolutions of the island of Reil, the external capsule being a correspondingly thin layer of white fibres on the inner side of the claustrum.

(*a*) *External Capsule and Claustrum.*—It has long been known that hæmorrhage to a very large amount is apt to occur between the external capsule and the external surface of the lenticular nucleus; and Charcot says that the artery here in fault—one of the lenticula—striated arteries arising from the middle cerebral—is so frequently affected by sclerosis leading to the formation of miliary aneurisms, with subsequent rupture and hæmorrhage, that it almost seems to deserve the name of "cerebral-hæmorrhage-artery." More than forty years ago Gendrin had already called attention to the fact of hæmorrhage occurring more outside than inside the grey nuclei of the corpus striatum; and his observations have since then been extended chiefly by Bouchard, Bourneville, and Duret, and in this country by Dr. Broadbent,[2] who has shown that the effused blood in its further course through the brain-substance performs a kind of natural dissection in the direction of the least resistance. Dr. Broadbent's cases, however valuable in themselves, cannot by any means be taken to afford any information concerning the special functions of the external capsule and claustrum, as has been assumed, for in none of them was the lesion limited to these structures; on the contrary, it was always found to extend to distant parts, generally ploughing

[1] 'Brain,' Part X. p. 147.

[2] On Ingravescent Apoplexy. 'Medico-Chirurgical Transactions,' vol. lxix. p. 335. 1876.

up the hemisphere more or less, and bursting into the lateral ventricle. This great extent of the hæmorrhage quite unfits these cases for being utilised for localisation, strictly so called, as for that only limited lesions are suitable. An additional reason for our not making such use of these cases is that all the patients died shortly after the attack. One of them, indeed, survived for three weeks and a half, but the others were carried off within some hours, or, at most, a few days after the seizure. Now recent cases, more especially of hæmorrhage, are most deceptive if employed for the purpose of localisation, inasmuch as at an early period of the disease the symptoms are not exclusively owing to destruction of tissue, but also very greatly to pressure of the clot on neighbouring parts, whereby the circulation in them is altered, and function inhibited. As the clot contracts, these latter symptoms gradually disappear; and about six or eight weeks after the attack those which remain are probably mainly owing to destruction, and then fit for diagnostic purposes. Dr. Broadbent has, in the paper just referred to (p. 358), called special attention to the circumstance that he found hemianæsthesia accompanying the paralysis, not only where the thalamus or the posterior portion of the internal capsule was damaged, but where the hæmorrhage was too far forward for either occurrence. This has led him to think that the French school has referred hemianæsthesia too exclusively to a lesion of the posterior part of the internal capsule, and that that symptom may also be induced by lesion elsewhere. It will be seen from the above considerations, however, that this conclusion as to the causation of hemianæsthesia is not justified by the cases on which Dr. Broadbent has relied for his argument. Had his patients survived the lesion a little longer, the hemianæsthesia might have gradually disappeared, as is not uncommonly seen in similar cases, showing that that symptom was owing not to destruction, but to temporary inhibition merely.

Hæmorrhage localised in the external capsule and claustrum is very rare; and softening limited to these parts does not occur. Temporary crossed hemiplegia would appear to be the symptom corresponding to the above lesion; yet there may be no morbid sign whatever from the commencement, and

it therefore seems impossible to diagnose disease of these structures.

(*b*) *Linsenkern, lenticular nucleus.*—Small areas of softening in this region may remain latent during life; but if the larger part of the nucleus be affected, opposite hemiplegia is the result, which is generally incomplete in character, and transitory in appearance. The recovery of function which appears to be the rule in such cases, is probably owing to compensation being effected by means of the uninjured portions of the linsenkern itself, or of the caudate nucleus. In some of these cases nothing but paralysis of the lower branches of the portio dura has been observed. Hæmorrhage seems never to be strictly limited to the linsenkern, but always to involve portions at least of the internal capsule at the same time; while tumours have been found involving symmetrically the nuclei of both sides, and yet had been totally unsuspected during life (Rondot, Fürstner).

(*c*) *Schwanzkern, caudate nucleus.*—Lesions affecting this ganglion in its entire extent, without encroaching upon other neighbouring structures, have not yet been observed, which is no doubt owing to the peculiar distribution of blood-vessels in this part. Its head alone has occasionally been found softened where there had been no symptoms during life exciting suspicion of such an occurrence. Where portions of the internal capsule have suffered in company with the schwanzkern, crossed incomplete and transitory hemiplegia has been the result. From all this it appears that we are unable to distinguish during life a lesion in one grey nucleus from a similar one in the other; and that, where hemiplegia remains permanent after an apoplectic seizure, the lesion cannot be limited to either of the grey nuclei of the corpus striatum.

(*d*) *Internal Capsule.*—It has been stated that hæmorrhage into, or softening of, the white medullary matter of the internal capsule always causes permanent and incurable crossed hemiplegia; but such is not the case. Here, as elsewhere, the extent of the area of disease is of the greatest consequence, for small lesions may exist even in this important locality without leading to striking symptoms during life. Nevertheless it is a fact that, in the vast majority of cases, somewhat extensive

destruction of this part is followed by crossed hemiplegia of the portio dura and the extremities. The conducting paths for the portio dura and the extremities are probably quite distinct within the internal capsule, as cases are on record where the extremities suffered exclusively, and others where the lower branches of the portio dura suffered exclusively, from disease in this region. The hypoglossus is not so commonly involved as the portio dura; indeed, the tongue appears sometimes quite straight, even in the commencement, and there may be no difficulty of articulation. In other cases the tip of the tongue deviates, and there is dysarthria or anarthria, such as we see it in medullary or pontine lesions. These symptoms, however, frequently vanish in the further progress of the case, from which it may be concluded that they were owing not to destruction, but to inhibition of the function of the cortical or subcortical centres of the hypoglossus, by pressure of the clot. As this latter contracts, the pressure is relieved, and function resumed to a proportionate extent.

That hemiplegia should be more or less permanent from disease of the internal capsule, and temporary from affection of the grey nuclei, is explained by considering the fibres of this capsule as centrifugal in their action, intended to transmit to the extremities and the portio dura the motor influence generated in the cineritious substance of the hemispheres as well as in the caudate and lenticular nucleus. While, therefore, lesion of the lenticular nucleus, for instance, will not prevent transmission of power from the caudate nucleus and the cortex of the brain, destruction of the internal capsule puts an impediment in the way of conduction of any motor power, from whatever source generated. It would therefore follow that only regeneration of nervous fibres in the capsule could, by re-establishing conduction between the centre and the periphery, lead to disappearance of the paralysis in such cases; and this appears, unfortunately, to be a very rare occurrence.

The localisation of the conducting paths for motion and sensation in the different portions of the internal capsule constitutes one of the most brilliant discoveries in this chapter of pathology. According to Charcot and Flechsig, lesions of the internal capsule alone, without any participation of the grey nuclei,

will cause permanent hemiplegia with late rigidity, without permanent affection of sensibility, if limited to the two anterior thirds of the capsule, where this separates the anterior end of the linsenkern from the head of the schwanzkern; while, on the other hand, destruction of the posterior third of the posterior segment of the internal capsule, where this lies between the thalamus opticus and the posterior end of the linsenkern, will cause crossed hemianæsthesia without motor paralysis.

Hemianæsthesia from disease of the internal capsule may be complete and permanent. It then affects not only the entire skin of one half of the body from vertex to toe, but also the mucous membranes of the eye, nose, mouth and tongue, the external auditory meatus, one half of the anus, glans penis, or vagina, as well as the special senses of sight (with negative ophthalmoscopic appearances), of hearing, smell and taste. In other cases it is incomplete, affecting only the skin, and leaving the mucous membranes and special senses intact; or slight in degree, so that sensation is only impaired, but not lost. Where it is complete, the muscular sense and electro-muscular sensibility are likewise absent, so that, while the nerves and muscles respond well to faradisation and galvanisation, no sensation is excited by the electro-muscular contraction; while tendon-reflex excitability is unaffected. In some cases hemianæsthesia disappears after a time, showing that it was not owing to destruction, but to inhibition of function; and similarly in a case where there were at first both hemiplegia and hemianæsthesia, the latter may remain and the former gradually disappear, showing that there was an inhibitory lesion in the anterior, and a destructive one in the posterior part of the capsule.

It is perhaps well to remark that hemianæsthesia of the skin may also occur in lesions of the mesocephale (pons, crus cerebri) and of the corona radiata, so that it cannot be considered as strictly characteristic of internal capsule disease.

Lesions of the *centrum ovale* are as yet little studied, and their symptoms are so manifold in character that in most instances in which they occur, an accurate diagnosis cannot be made during life. Comparatively large portions of this connecting link between the central ganglia and the cortex

may be destroyed by softening or hæmorrhage, without giving rise to any symptoms at all. This appears to be more particularly the case where the occipital, parietal, sphenoidal, and anterior and middle frontal portions are affected. On the other hand, disease in the posterior frontal and posterior central part causes the typical symptoms of corpus striatum paralysis, *i.e.* opposite hemiplegia with paralysis of the lower branches of the portio dura, and sometimes of the hypoglossus. Where the left hemisphere is the seat of it there may also be aphasia, which we are at present unable to distinguish from aphasia owing to destruction of Broca's convolution; and dysarthria which resembles the same symptom from disease of the pons Varolii, medulla, &c. The lesion of aphasia is in the posterior frontal part, close by the third left frontal convolution, and is probably owing to disruption of what Meynert has called the "system of association." Where a small area is affected, there may be only monoplegia. Early and late rigidity in the paralysed muscles is also sometimes found. Tumours invading the centrum ovale may give rise to the general symptoms of increased intracranial pressure, such as vomiting, optic neuritis, headache and epileptiform seizures, without the presence of any regional phenomena enabling us to localise the seat of the disease. It is also worthy of note that destruction of the occipital portion of the centrum ovale has been found associated with acute bedsores on the opposite side.

By lesions of the *cortical substance* of the hemispheres we understand not only those which affect the cineritious substance itself, but also the white medullary matter immediately underlying the same. Of all cerebral convolutions the third left frontal has, since Broca's observations, received the largest share of attention; and a number of typical cases in which there was limited destruction of the same, have rendered its intimate relation to the production of intelligent language absolutely certain. Yet a number of questions connected with this point still await solution. Is this the only centre for language, or are there other portions of the brain which have a close and constant relation to that faculty? What is the part played by the island of Reil and the first left temporal

convolution? How are we to explain some undoubted cases of destructive lesions, not only in the left, but in both third frontal convolutions, in which there was no aphasia? Have the parietal lobes any relation to the faculty of intelligent language, &c.?

As at the present day any opposition to the main features of Broca's theory proceeds only from those who are wilfully blind or disingenuous, the least that the adherents of that doctrine have a right to ask for from their opponents is a thoroughly complete description of the cases brought forward, with the view of disproving the same. Thus it is indispensable to know whether the subjects have been right or left-handed during life; for it is an established fact that, where there is congenital or acquired deficiency of the left hemisphere, the right side of the brain will be trained during infancy for speech, and for the more complex movements of the upper extremity. The omission of this particular renders such cases as those of Simpson, Christison and others, entirely devoid of value. Then, again, it is now well understood that part of the third convolution may be destroyed, and yet its function not be seriously interfered with, if only that portion of it in contact with the operculum—which is situated at the junction of the two branches of the fissure of Sylvius, and covers the convolution of the insula—has remained intact. It is therefore an important reservation to state that language is, in general, *bound to the opercular portion of the third frontal convolution.* For other cases which are apparently contradictory, the bilateral function of the parts in question offers a ready solution. With all these reservations, however, a few cases remain which would seem to show that the function which is in the immense majority of cases bound to the third left frontal may occasionally be undertaken by the insula of Reil, either alone, or in connection with the first left temporal convolution and perhaps the gyrus angularis. A somewhat constant relation of the first left temporal to Kussmaul's "verbal deafness" seems to have been established. Patients of this kind appear either deaf or insane, although they are neither, because they are unable to realise the meaning of spoken words; and this affection is in some instances combined with aphasia, agraphia,

and alexia. It is an interesting fact that in one of Kussmaul's cases verbal deafness was associated with disease in the right first temporal in a man who had been left-handed during life, and in whom therefore congenital deficiency of the left hemisphere could be assumed.

Next in importance to the questions connected with the third frontal and the first temporal is the occurrence of motor paralysis from disease of the convolutions bordering the fissure of Rolando, viz. the anterior and posterior central, and the paracentral lobule. Lussana and Lemoigne have entirely failed to shake the large array of facts which prove such a connection; yet numerous points in this matter are not as yet cleared up. If we accept, for instance, the topography of centres for the tongue, portio dura, and extremities, as given by Charcot and Pitres, it is difficult to explain the occurrence of cases of hemiplegia resembling that which is caused by internal capsule hæmorrhage, and coming on without disease involving the entire region of Rolando, the lesion being limited to a comparatively small area in it. Again, the dissociated or piecemeal appearance of paralysis is not absolutely distinctive for cortical lesions, for occasionally loss of motor power may come on suddenly together with loss of consciousness, in disease of the central convolutions; while it may appear piecemeal, and without affecting the sensorium, in lesions of the pons Varolii, the crus, and the centrum ovale.

The occurrence of signs of motor iritation which is generally believed to be characteristic of lesions in Rolando's region, likewise raises questions which have not as yet been satisfactorily answered. Leaving out of view the presence of late rigidity owing to secondary sclerosis in the pyramidal strands, which may follow cortical lesions just as well as corresponding affections in the centrum ovale, the internal capsule, &c., we are confronted with localised clonic or tonic spasms, and with cortical or Jacksonian epilepsy. Why do these symptoms occur in one set of cases, and not in another apparently exactly similar one? How are they produced? And what is the reason that neither the kind of disease—whether tumour, softening or hæmorrhage—nor its seat in one or the other of the three convolutions in question, nor its invasion of the

cineritious or the underlying medullary matter, seem to have any influence in the production of these phenomena. That their presence or absence follows a definite law cannot be doubted; but much careful clinical as well as pathological work will have to be done before this law can be formulated.

The question of treatment of localised brain-lesions has been generally lost sight of by those most eager to investigate their symptoms and pathology; yet this must for all time to come be the chief point of interest for the practical physician. I will in this place not speak of the attempts which have been made to arrive by trephining at the seat of the disease, as I have elsewhere expressed my disbelief in the future of cerebral surgery.[1] A subtler agent than the surgeon's knife or trephine is wanted for this branch of therapeutics. The chief practical points I wish to submit to the attention of neurologists in this place are two, viz. 1st, the expediency of treating tumours of the brain as if they were syphilitic in origin, even where this cannot be demonstrated; and 2nd, utilisation of the catalytic effects of the constant voltaic current, in certain destructive as well as irritative lesions, directed locally to those portions of the brain where we suspect the seat of the mischief, in accordance with the teachings of pathology and with Ferrier's physiological topography of the cerebral cortex.

I will now relate a case in illustration of my first point, viz., the advisability of antisyphilitic treatment for tumours of the brain:[2]—

Case 1. *Tumour in the Right Cerebral Hemisphere.*—A merchant, aged 32, married and father of two healthy children, consulted me in April 1878. He had always been in good health until about six months ago, when apparently without any particular cause he began to be seized occasionally with attacks of giddiness and vomiting. This was believed to be owing to congestion of the liver, and treated as such. Headache supervened after a time, his sleep became disturbed, and he appeared sometimes delirious at night. The appetite began

[1] 'Diseases of the Nervous System,' vol. i. p. 45.

[2] A similar case was recently brought before the Medical Society of London, and afterwards published in the 'British Medical Journal,' by Dr. Hughlings-Jackson.

to fail next, and he could not be induced to eat anything substantial. He generally brought up almost directly what little he took. When I first saw him, he was so much emaciated that his friends believed him to be consumptive. He had a sallow complexion, and a careworn countenance. He complained of persistent headache, which was generally of a dull aching character, but sometimes became acute and throbbing, and "made him feel quite silly;" of attacks of giddiness which seemed to take him off his feet, so that he felt as if he was floating in the air; and of greatly impaired memory and application. He had not been to his office for some weeks past, as he was incapacitated from fixing his attention on those subjects which demanded it, and even to look at a column of figures quite upset him. There was optic neuritis in both eyes, with slight extravasation of blood, turgid veins, and contracted arteries; he could not read No. 2 Jaeger. No other cranial nerves were affected. He had lost power in the left side of the body; his left hand was nearly useless, and showed a grasping power of 25 on the dynamometer, while the right showed 120. The left leg was very feeble, and the left foot apt to drag on the ground, so that the patient avoided walking as much as possible; but there was no reeling gait, such as is seen in cerebellar disease. There was no atrophy in the paretic muscles, which responded equally well to faradisation and galvanisation as those of the right side. Sensation was not impaired, the reflex action of the skin and the mucous membranes normal, that of the tendons appeared somewhat increased. The urine contained an excess of phosphates, but no sugar, albumen, or excess of urea. The action of the bladder and rectum was normal, and there was no sign of disease of any of the other viscera.

Tumour in the right cerebral hemisphere was diagnosed, probably about the ganglionic masses of the corpus striatum and thalamus opticus. The patient was severely questioned about any syphilitic antecedents, but denied ever having had any primary or secondary disease. He had always been extremely steady in his habits, and had never had connexion before marriage. His wife had never had any miscarriages, and she as well as the children appeared perfectly healthy.

I nevertheless instituted an anti-syphilitic treatment, as this appeared to me the only chance of benefiting the patient. One-sixteenth grain of perchloride of mercury was given together with ten grains of iodide of potassium twice a day. The effect of this medication was almost magical. The vomiting ceased after the first dose, and never reappeared at any subsequent time. The appetite began to revive, the headache became less intense, and there was a gradual and steady amelioration in all the symptoms from which the patient had suffered. About a fortnight after commencing the treatment he returned to business, in which he took renewed interest; and rather more than three months afterwards he appeared quite well. No other medicine had been given except cod-liver oil, of which the patient took two tablespoonfuls daily from the third week, and which was continued altogether for six months. The perchloride of mercury had to be occasionally withdrawn for some days on account of affecting the gums too much; but it was always noticed that there was not nearly the same improvement in the more important symptoms when potassic iodide alone was given; and that the patient progressed much more quickly when the perchloride was resumed.

I now proceed to give the details of some cases in which localised lesions of certain portions of the brain, other than tumour, were diagnosed, and in which the catalytic effects of the constant current proved of service.

Case 2. *Diabetes insipidus, Galvanisation of the Medulla.*—A gentleman, aged 37, single, consulted me on July 27, 1880. He had spent many years in the tropics, and had suffered from persistent diarrhœa, which nothing would arrest. This ultimately brought on a state of complete cerebral exhaustion, and he became quite unable to attend to his occupations. The most troublesome amongst the numerous symptoms from which he suffered, however, was that of polyuria, which was so bad as to exclude him altogether from society. When in company, he could hardly sit still for a quarter of an hour without experiencing a most pressing desire to empty his bladder; and this annoyance had led him to adopt a solitary mode of life. The average quantity of urine which he passed

during the day amounted to ninety ounces, but often much more. It was feebly acid, had a low specific gravity, and contained nothing abnormal. As he had taken gallons of physic without the slightest relief, he wished to ascertain from me whether any mode of applying galvanism might be expected to be of service to him.

I have elsewhere[1] given my reasons for considering diabetes insipidus an affection of the medulla, possibly in connection with the middle lobe of the cerebellum, and for looking upon any ultimate organic lesions in the kidneys as secondary, and owing to the maceration of renal tissue through excessive and long-continued diuresis. Proceeding from this view, I directed the current of fifteen cells of Becker-Muirhead's battery to the back of the head, taking care that the medulla received alternately the influence of the anode and cathode, and regulating the finer degrees of voltaic power by the rheostat. The application lasted altogether six minutes, and was entirely painless.

The patient came to see me again a week afterwards, and informed me that the result had been completely successful. The quantity of urine passed during the day had, after the application, fallen to thirty ounces; and while formerly he had often had had incessant calls to pass his water, he had since then only been obliged to pass it three times a day. I regret to say that I had not the opportunity of examining the urine more carefully, either before or after the application of electricity, and that I have since lost sight of the patient, so that I do not know whether the relief has continued. That the change which occurred so suddenly was really owing to the use of the galvanism seems obvious, as no medicine had been given, and the patient had not adopted any alteration in his diet or general regimen. The derangement of the medulla, and possibly of the middle lobe of the cerebellum, which gave rise to the polyuria, must have been slight, and may have been anæmia, or a degree of passive hyperæmia. It was, however, sufficient to render the patient thoroughly miserable, and might perhaps in course of time have developed into more serious organic changes in the organ.

[1] 'Medical Times and Gazette,' November 27, 1880.

CASE 3. *Melancholia; Galvanisation of the Occipital Lobes.*—A married woman, aged 28, had her first confinement in July 1877. She had until that time been habitually in the enjoyment of excellent health, and been bright and cheerful in her manner; but soon afterwards a complete change in her disposition was observable. She took a dislike to her husband, seemed to lose all interest in the affairs of her home, and became sullen and morose. She refused to go out, and would sit all day long in a corner of her room, doing nothing. If she conversed at all, it was on religious subjects, in which she had previously shown a very moderate interest only; and the only book she would now read was the Bible. She had a settled conviction that she could not be saved, and was doomed to go to hell. When her friends attempted to reason with her, she became only more rigidly fixed in her ideas, and appeared to resent every interference. I first saw her in March 1878. She had then the expression of a settled frown on her face; the pupils were large; the tongue furred; speech slow and measured. I had much difficulty in inducing her to answer my questions, which she evidently thought impertinent. The appetite was poor, the bowels confined, the catamenia regular. She had never nursed her child. Phosphorus, strychnia, and other nerve-tonics were given, but did not afford any relief. She had been taken away from her home for a complete change, but this had done no good at all. After two months of ineffectual medical treatment and regimen, I proposed the application of the constant current. This was directed to the occipital lobes, with voltaic alternatives, for five minutes at a time. An improvement soon became manifest, and when the treatment was discontinued after twenty applications, the whole aspect of the case appeared altered. The patient was still more reserved and silent than she had originally been, but she was cheerful, and took more interest in the affairs of daily life. She felt more inclined to take exercise, her appetite had improved, and her ideas on religious subjects had undergone great modifications.

CASE 4. *Auditory delusions; Galvanisation of the Temporo-sphenoidal Convolutions.*—A gentleman, aged 38, had for some

years past suffered from epileptic seizures, for which he consulted me in February 1879. His memory had become impaired, but his intellect and judgment were good. One morning he came to me in great apparent distress, expressing an apprehension that he was going to become insane, as for some days past he had been subject to delusions, which had come on without any perceptible cause. He constantly heard voices behind his back, calling him names and speaking about his pecuniary affairs and his state of health; and he appeared quite worn out by anxiety on this account. Looking upon this symptom as denoting hyperæsthesia of Ferrier's auditory centres in the superior temporo-sphenoidal convolutions, I applied the current to those portions of the skull corresponding to these parts, for five minutes consecutively. This gave immediate and thorough relief, as the delusion had completely vanished at the end of the application, and did not return on any future occasion.

Case 5. *Corpus Striatum Hæmorrhage.*—A widow, aged 60, had an attack of apoplexy on January 30, 1879. The first symptom was giddiness, which passed within half an hour into complete insensibility. There was involuntary evacuation of the urine and fæces. The patient remained unconscious for about three hours, after which she gradually came to, and appeared next day to be almost herself again, but that there was hemiplegia of the left side. I first saw her ten days after the attack, and found all the symptoms of recent corpus striatum hæmorrhage. There was paralysis of the lower branches of the portio dura, which became plainly visible on laughing and on attempting to show her teeth; she had difficulty in swallowing solids; the tongue appeared bluish, congested and deviated to the paralysed side. Articulation was somewhat indistinct, but the patient had no difficulty in expressing herself. There was no headache or giddiness, but some tenderness on percussion over the right hemisphere. There was no conjugate deviation of the head and eyes. The left arm was completely paralysed, and appeared a piece of inanimate matter, not the slightest voluntary movement being possible in any part of it. The leg, which had at first been as much paralysed as the arm, had regained a slight degree of

motive power, more especially in the knee-joint and the toes. There was also a degree of paralysis in the muscles of the body, for the patient was unable to shift her position in bed, nor could she raise herself from the lying to the sitting posture without assistance. Sensation was normal everywhere; reflex action of the skin and tendons not increased; no ankle-clonus. The faradic excitability of the paralysed muscles was normal. Respiration on both sides of the chest tolerably equal; pulse 90, full, firm; temperature 98·5°; no acute bed-sore anywhere. The appetite was tolerably good, and urinary and alvine secretion satisfactory; she slept pretty well, and was in good spirits. I prescribed a phosphorus perle to be taken twice a day, and recommended the application of the continuous current as soon as ankle-clonus should become manifest. This was first noticed on the twenty-seventh day of the disease. The current was now applied to the right hemisphere, the cathode and anode being alternately directed to the crown of the head and occiput, after which the stream was sent transversely through the brain, from one mastoid process to another. Ten cells of Weiss's Leclanché were used, for eight minutes altogether. This was done on alternate days four times, after which the paresis of the muscles of the body had completely disappeared, the patient being able to sit up in bed and change her position without any trouble. The mobility of the leg had also improved, but there was now slight rigidity in the hamstring muscles. The shoulder appeared more movable, but some rigidity in the biceps and flexor communis digitorum was noticed. The galvanic application to the hemisphere was repeated, and a gentle application to the limbs combined with it. After another week of this treatment the patient had almost entirely recovered the use of the leg, and was able not only to walk about in her room, but also to go up and downstairs without assistance. The rigidity in the muscles of the arm had meanwhile become more marked, but there was also greater mobility, not only in the shoulder but also in the elbow and fingers, while the wrist remained quite motionless. The treatment was pursued with some interruptions for four months. The patient was then able to walk three or four miles at a time without the least

difficulty; indeed all traces of paralysis, as well as rigidity in the lower extremity had disappeared. The arm and hand had not made quite the same progress, but the patient was able to use the hand for dressing and eating; and, although several muscles were still paretic and stiff, she was not nearly so dependent upon others as she had expected to be.

Improvement may follow the systematic use of the voltaic current to the suffering hemisphere even years after the occurrence of the stroke, and when we should expect that secondary sclerosis of the parts below the lesion would render all therapeutical efforts futile. This is shown by the following case:—

Case 6. *Softening of the Left Hemisphere from Embolism of the Middle Cerebral Artery.*—A single lady, aged 40, had had a stroke which took away her speech and the use of the right side of the body, in summer 1868. I first saw her in July 1878, that is, ten years after the occurrence of the attack. The early history of the case could not be very fully ascertained, but there was little doubt from the symptoms present that she had suffered from embolism of the left Sylvian artery, with consecutive necro-biotic softening in the sphere of that vessel. There was now distinct aphasia, the patient being only able to say "yes," "no," and a few other words, such as "doctor," "my hand," &c.; yet she understood everything that was said to her, could read the paper, and express her wants by pantomime. She behaved quite rationally, and her intellect seemed to be good. There was slight affection of the lower facial muscles; the tongue was thin, and could only be protruded a very little way, but was not deviated. The whole right arm was in a state of paralytic rigidity, and utterly useless. The wrist could neither be raised nor pronated nor supinated, and the fingers were so powerfully flexed that the nails touched the skin. The hamstring muscles of the thigh were likewise contracted, and the patient, although able to walk, was very lame. There was no wasting of muscles, which responded well to faradisation. Ankle-clonus was well marked. The general health of the patient was good, except that she had three years ago lost the catamenia, since when she suffered from periodical headaches.

A similar treatment was resorted to in this case as in the

above, and continued for six months. At the end of that time the rigidity of the leg had almost entirely disappeared, and the patient walked with great ease. There was considerable amelioration in the use of the right hand, which could be used for dressing, eating, needlework, embroidery, etc., but not for writing, which the patient had learnt to do with the left hand. The most satisfactory feature, however, was the improvement in her language. The patient had regained the command of a large number of words which she had lost before; she was able to read aloud and to converse, and on many occasions pronounced long sentences with tolerable fluency. She was however easily flurried, more especially in the presence of strangers, and then would begin to stammer and to lose the thread of the conversation; she would, however, recover herself after a few minutes, and then speak comparatively well again. The catamenia reappeared after the galvanic treatment had been followed for some weeks; and, although they did not again become thoroughly regular, they continued at intervals varying from six to ten weeks; and she lost the periodical headaches to which she had been subject before.

The precise mode of action of the constant current in destructive brain-lesions will probably not be understood until a number of cases which have been treated in a similar manner have been thoroughly investigated post-mortem. Is there true regeneration of nerve-fibres in the internal capsule under such circumstances, and can secondary sclerosis below the lesion be repaired when the conduction between the cortex and the periphery becomes, at least to some extent, re-established? That repair of destroyed nerve-fibres may occur has been shown not only by experiments on animals, but also by cases of neurectomy for trigeminal neuralgia, in which anæsthesia owing to loss of nerve-substance (amounting in some cases to as much as three-quarters of an inch) was recovered from, and the paroxysmal pain, which had disappeared after the operation, returned in the same sphere after a time. This points in the most unmistakable manner to re-established conduction. Can part of the internal capsule be repaired in a similar manner? Or are the therapeutical results of galvanic applications owing to a more complete removal of the causes

of inhibition, and to the induction of vicarious function on the part of neighbouring structures? These questions have a great theoretical as well as practical interest, and demand the serious attention of neurologists. In the meantime not too much stress can be laid upon the importance of treating such lesions at an early stage, and before secondary sclerosis has been fully established. With regard to this point the occurrence of ankle-clonus after an apoplectic or paralytic seizure should be carefully watched for, as it is a diagnostic sign of considerable value leading us to predict the probably speedy appearance of late rigidity in the paralysed muscles; and as soon as this symptom has made its appearance, no time should be lost in resorting to the use of electricity.

OBSERVATIONS ON CERTAIN OPTICAL ILLUSIONS OF MOTION.

BY CHARLES S. W. COBBOLD, M.D.

Assistant Medical Officer, Colney Hatch Asylum.

IT is proposed in the present paper to consider some of the arguments and conclusions recently advanced by Professor Silvanus Thompson upon this subject,[1] and to offer additional suggestions. In order as far as possible to avoid repetition, the present writer will assume that his readers are familiar with the contents of Professor Thompson's article. By the courtesy of that gentleman, the figures which illustrated his paper are here reproduced for convenient reference.

First, as to the *law of subjective complementary motion*, which Professor Thompson enunciates. It appears to afford a satisfactory explanation—the only one which can at present be offered—of certain particular optical illusions of motion not explicable by any other theory. The new law is as follows:—"The retina ceases to perceive as a motion a steady succession of images that pass over a particular region for a sufficient time to induce fatigue; and on a portion of the retina so affected, the image of a body not in motion appears by contrast to be moving in a complementary direction."[2] By this law the following illusions are capable of explanation: 1. After looking from a rapidly moving railway-carriage at objects lying beside the line, as they pass before the eyes, let the eyes be closed suddenly, an apparent motion in the opposite sense will at once be perceived, undistinguishable forms and patches of light seeming to rush past the blank field. 2. After looking for some time at a waterfall, and then at the rocks adjacent, the rocky surface may be seen to appear as if in motion upwards. 3. The appearance of reversed rotation which

[1] 'BRAIN,' Part XI., October, 1880. [2] L. c., Vol. III. page 296.

is seen after looking for some time at a slowly revolving disc divided into black and white sectors (Fig. 3). 4. The apparent expansion of surrounding objects after looking at the end of a retreating railway-carriage. 5. The apparent convergence to, and divergence from, a given centre, of objects looked at after the eye has been fatigued by regarding a revolving disc of black and white spirals (Fig. 4). As regards these illusions and others of a similar nature, the "muscular slipping" theory fails entirely, but Professor Thompson's new law is perfectly satisfactory.

The apparent rotation of the concentric circles (Fig. 1)

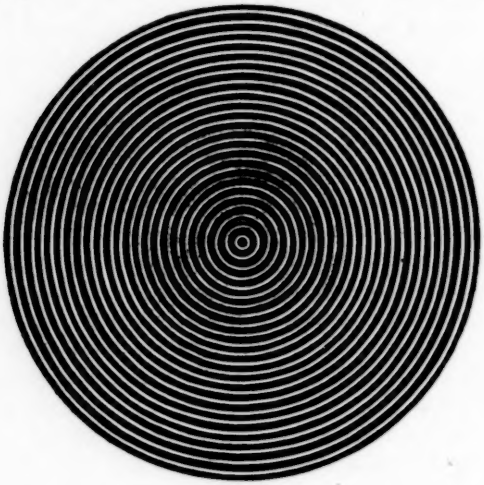

FIG. 1.

cannot, however, be explained by this law. Professor Thompson himself says that "the persistence of visual impressions has a good deal to do with the production of this illusion," but he does not explain in what way. The following appears to be the true explanation of the manner in which the persistence of visual impressions gives rise to the appearance of rotation of "strobic circles" to which a "rinsing" motion is imparted; and it is believed that by it all the facts of this curious illusion can be explained.

If the circles be "rotated" as slowly as possible, it may

be noticed that the appearance of rotation is due to the seeming revolution of apparent radii or sectors. The whole disc appears divided into four sectors—two narrow ones, which are opposite to, and in a line with, one another, and in which the black and white bands are distinctly defined—and two much wider ones, in which the black and white rings become somewhat lost in one another, causing a blurred appearance. These sectors may be best seen by moving the circles somewhat rapidly upwards and downwards, that is, vertically, without lateral motion. The clearly-defined, narrow sectors will then be seen extending horizontally on either side of the centre, while the upper and lower portions of the disc appear somewhat indistinct, brownish, and blurred. If the disc be oscillated laterally, without vertical motion, the clear sectors at once assume a vertical direction; they are, in fact, always perpendicular to the direction of motion.

The explanation of this appearance is obviously to be sought in the persistence of retinal impressions. During vertical motion, the images of the more or less horizontal black and white bands which occupy the upper and lower parts of the disc are constantly replacing one another upon the retina, each becoming confused with the impression immediately preceding it, and thus producing the blurred appearance noticed. The reverse is the case in the narrow lateral sectors, in which the black and white bands are practically vertical, and coincide with the direction of motion; their images consequently do not displace one another upon the retina, but remain clearly defined and unaffected by the vertical movements. It is very difficult to produce, with the unaided hand, to-and-fro movements which shall be either purely vertical or purely horizontal; the result of this is that, in trying this experiment, the clear sectors do not remain absolutely fixed in position, but oscillate in the sense of the imperfections of movement, reminding one much of the behaviour of the magnetic needle when shaken or jolted.

In the rinsing movement, which produces apparent rotation of the circles, the disc is moved in all directions successively during each revolution: the clear sectors being, as we have seen, always perpendicular to the direction of motion, must,

therefore, also move in all directions successively, following the direction of motion, though constantly at right-angles to it. An appearance of revolving radii, similar to that produced by the spokes of a rapidly revolving wheel, is thus brought about. It is almost needless to point out that if a series of perfectly concentric black and white circles were set revolving, no such appearance as that now described (of revolving radii or sectors) would be produced; indeed, if the bands were of unvarying width, and with very clearly defined borders, it might be difficult to discern, apart from other evidence, whether revolution were really taking place or not.

It will be found that several other curious facts connected with this illusion are easily explicable in accordance with the explanation just offered. The reason why the circles appear to revolve with the same angular velocity as that imparted is at once obvious.

"It is found that for increasing distances from the eye the concentric rings must be made wider if the illusion is to succeed; there being apparently one particular magnitude of their images on the retinæ, which favours the production of the illusion."[1] This quite accords with the explanation offered; the black and white bands must be large enough to be easily and clearly distinguishable by the eye when they are in the clear sectors of the disc; but they must not be so large as to interfere with the necessary confusion of the images received upon the retina from the greater part of the disc's surface. If the bands were of a certain magnitude, it would be competent for the eye to fix itself on one particular band and follow its movements in every direction, in that case there would be no rapid sequence of images passing over any portion of retinal surface, and no illusion would be produced. The happy medium in the size of the bands, which is necessary to the success of the illusion, must obviously, in accordance with this explanation, vary with their distance from the eye.

If two sets of circles printed upon one card be "rotated," those at which the eye is not looking revolve most vigorously. This is another way of saying that the circles rotate best when their

[1] L. c. p. 293.

images fall upon parts of the retina which are somewhat removed from its centre. The circles always rotate best if the eye be fixed on some point a little outside them. My explanation of this fact is that, the more peripheral parts of the retina being less sensitive than the central, the psychological element plays a larger part in the interpretation of the impressions received, and, being supplied with more imperfect data, is more liable to arrive at erroneous conclusions as to the mode of causation of the images perceived. The physical impressions being defective, what wonder if the psychic faculties fail to attribute them to their true origin?

Again, "on stopping the 'rinsing motion' suddenly, there appears to be, for an instant, a reverse motion."[1] For this, I believe, there are two causes; the first and most important is to be found in Professor Thompson's law of subjective complementary motion. The second cause is that the hand, in trying suddenly to stop the circular movements, often involuntarily commences a revolution in the opposite direction, thus causing the clear sectors also to reverse the direction of their movements for an instant.

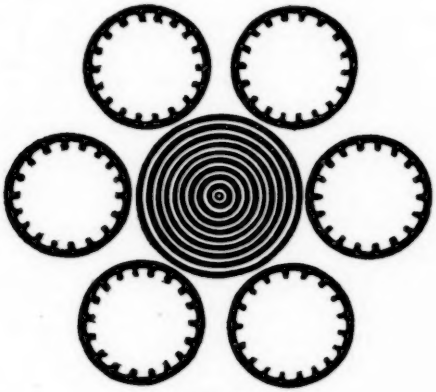

Fig. 2.

Coming now to the toothed wheels (Fig. 2), I believe that their apparent rotation during "rinsing movements" must

[1] L. c. p. 293.

be explained by yet another theory. The mere fact that the wheels only move through one tooth while the circles make a complete revolution, and in a contrary direction, is of itself sufficient to indicate that the two illusions are essentially distinct and susceptible of different explanations. It seems to me that it is here that the "muscular slipping" theory, or something very like it, comes in. It is evident that the eye, from some cause or other, once in each revolution, mistakes the image of each tooth for that of the one next adjacent; the wheel thus appears to have moved onwards through one tooth.

Adopting the "muscular slipping" theory, the following would be the sequence of events. The eye follows the movement of each tooth for a certain distance, but, not moving fast enough, it catches sight of, and slips back to, the next following tooth, and mistakes it for the original one. Of course, the persistence upon the retina of the original impression aids materially in bringing about the apparent coincidence of the two images.

The matter may perhaps be put more correctly thus: the eye, in order to avoid the oscillations necessary to follow each movement of the teeth, remains almost stationary, and passively accepts the image of the next tooth which appears near the position of the previous one, as a continuance of the original impression, which original impression has, in fact, persisted until, or nearly until, the new one takes its place.

The toothed wheels rotate far better when the eye is not directly fixed upon them; in fact, this is a matter of much greater importance with the toothed wheels than with the strobic circles. This explains why the wheels are so effective when arranged round concentric circles, as these latter divert the eye from regarding the teeth too minutely. It is possible, with some effort, to fix the attention for a time upon one single tooth, in such a way that the illusion cannot be caused by rinsing movements, unless these be more rapid than can be produced by hand.

It will be found that the disc of black and white spirals, shown in Fig. 4, may be made to "rotate" perfectly if the eye be kept fixed upon the centre of Fig. 3, while rinsing

movements, with a somewhat large radius of motion, are made. The great indistinctness of the images formed upon the peripheral parts of the retina, together with their persistence,

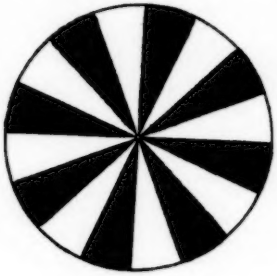

FIG. 3.

FIG. 4.

allows of each black spiral being in turn mistaken for the one next to it. In accordance with this view, the disc should rotate once for each three revolutions of imparted motion. This is apparently the case, but it is difficult to count the revolutions, as the illusion ceases immediately the eye is fixed upon the figure for that purpose. If the eye be kept fixed upon one of the spirals, the illusion cannot be produced; unless, indeed, the eye has become considerably fatigued.

Three distinct causes of optical illusions have now been noticed. First, that given in Professor Thompson's new law; little has been said concerning it, because it is fully explained in the paper in which it is enunciated. The other two, represented respectively by the illusions of the strobic circles and the toothed wheels, have been more fully considered. The correctness of the views here given, as concerning the particular phenomena attempted to be explained by them, in no way implies that optical illusions of motion are not produced in various other ways. I mention this because Professor Thompson rather appears, in his paper, to proceed upon the assumption that, because the muscular slipping theory can be proved not to apply in certain cases, it therefore does not apply in any.

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

BY BEVAN LEWIS, L.R.C.P. LOND.

Senior Assistant Medical Officer, West Riding Asylum.

(Continued from Vol. III., p. 515.)

III.

Œdematous Conditions of the Brain.

An œdematous state of the cerebral tissue accompanies all cases of softening of the brain, whether of inflammatory or non-inflammatory origin; but it is thought desirable to give an independent consideration to cerebral œdema, often the only morbid condition recognised by the naked eye in the brain of the chronically insane. As it is chiefly compensatory in its origin, it will be considered together with that compensatory effusion into the ventricles and beneath the membranes of the brain which so frequently accompanies it. If the cerebral tissue becomes much infiltrated with serum, it is liable to break down the texture of the brain. As before stated, this may proceed to a state of white softening, in which the brain tissue may become quite diffluent, such as we observe in the neighbourhood of the lateral ventricles in acute hydrocephalus and around inflammatory foci. The more frequent condition met with in the brain of the insane is that of a general moist condition of the white matter, which is soft, almost pasty, clings to the blade, and is apt to tear away in shreds of a dirty white hue. Such is the appearance in cases of senile wasting of the brain. The white medullary strands immediately bordering upon the lateral ventricles will usually be found most implicated, being here in closer contact with the serum contained in these cavities. The lesser

degrees of œdema are recognised by pitting of the tissue on pressure and a peculiar and notable brilliancy of the white substance when cut across, one of the most frequent appearances in the brain of the insane. The student must learn to recognise the *general moisture, swollen condition*, alterations in *consistence*, and alterations in appearance from a *brilliant to a dull dirty-white hue*, associated with varying degrees of œdema. If the freezing microtome be liberally used, he cannot fail to appreciate to the fullest extent the varying degrees of œdema of the cerebral tissue; and this not only because it proves the most troublesome obstacle to a free manipulation of delicate sections, but from the tendency of œdematous brain to freeze into a hard icy solid, differing much from the consistence of healthy brain when frozen. It is impossible to cut through such frozen brains except by a modified process to be alluded to further on. In the hardening processes, also, these brains undergo by dehydration enormous shrinking. The serous effusions beneath the arachnoid and into the ventricles, which have been alluded to as often associated with œdematous conditions of the brain, may occur as the result of—

a. Senile atrophy of the brain (compensatory).

b. Pressure by morbid products on the vessels, as in—

Tubercular meningitis.

Syphilitic disease of blood-vessels.

Tumours.

Abscesses of mid-lobe of cerebellum pressing on the venæ Galeni.

c. As a gradual accumulation from anæmia and other existing morbid states, as in chronic phthisis.

Where does the effusion of serum attending œdema of the brain occur?

a. Into sac of arachnoid, i. e., betwixt the cerebral layer of the arachnoid and the polished inner surface of the dura mater. On removal of the brain, the student will note this fact by examining the amount accumulated in the occipital fossæ above and below the tentorium.

b. Beneath the cerebral arachnoid. Observe how the latter is floated up by the subjacent fluid where this membrane bridges across the sulci.

c. In the meshes of the pia mater. Note its swollen gelatinous appearance from infiltration with serum.

d. Into the ventricles:—

1. More or less distending the lateral ventricles.
2. Stretching tense the thin arachnoid which extends between the cerebellum and medulla over the fourth ventricle.

Before discussing the subject of œdematous conditions of brain and compensatory effusions of serum, we would recommend the student to acquire information upon the following facts in all such cases:—

1. Condition of ependyma (lining membrane of ventricles), *a.* As to healthy aspect. *b.* Granular appearance and feel. *c.* Macerated aspect. *d.* Fibro-cartilaginous plates.
2. Turbidity or discoloration of serous fluid.
3. Specific gravity of serous fluid.
4. Reaction to litmus paper.
5. Specific gravity of the brain.

Augmented Consistence.—All cases of induration of the brain should induce the student to examine the morbid change in the texture as regards its *degree*, its *distribution*, and *nature*. The cases most likely to present themselves to his notice may be embraced under the following categories:—

1st. General augmented firmness of the whole brain from increase of its connective or neuroglia element.

2nd. Limited but extremely indurated patches or nodules due to morbid growths, especially the carcinomata.

3rd. Limited indurations of grey or white matter due to transformation of inflammatory products.

4th. Sclerosis disseminated throughout the brain or distributed down the motor strands.

The student will observe that the increase in consistence may vary from a scarcely appreciable degree, being due merely to a lessened quantity of water (Rokitansky) up to a condition of leathery callous consistence.

A few words upon the varieties of sclerosis met with in cerebro-spinal centres. First the student will become familiar with the general increased firmness of the brain from in-

crease of its neuroglia on examining the brain of many epileptics.

Superficial Scleroses.—He should also look for instances of *partial sclerosis of the grey cortex* occasionally to be seen in epileptics and imbeciles, and characterised by a peculiar shallow puckering of the surface of the gyri, which are somewhat indurated and have a cauliflower appearance.

Hypertrophy of Neuroglia.—Again he may meet with instances of hypertrophy of the brain, as it is termed, and which has been found due to extreme increase of the neuroglia around the medullary strands of the hemispheres. Such a case would present him with increase in *consistence, elasticity,* and *volume.* In all cases of general increased consistence it is imperative that the greatest attention should be paid to examination of the brain under the following heads :—

a. Volumetric measurement.

b. Absolute weight of the brain and its divisions.

c. Specific gravity of grey and white matter.

Cicatricial Formations.—The indurations resulting from inflammatory action will frequently attract attention. Such patches of callous tissue may surround foci of softening of the cortex due to plugging, or the site of old hæmorrhages. Cyst-like cavities, with hard fibroid walls, may thus be formed deep in the substance of the hemispheres. Sections through such structures should be made for microscopic examination, as they illustrate well the various stages of transformation of inflammatory products into callous cicatricial tissue.

Disseminated Sclerosis.—If in slicing a brain greyish nodules are found scattered irregularly through the white substance resembling the cineritious substance in colour, but becoming of a rosy hue on exposure and exuding a colourless fluid, the student has probably to deal with a case of disseminated sclerosis. Note in such a case the increased consistence of the sclerosed patches, their vascularity, irregular distribution through the medullary substance, and the comparative immunity from this lesion enjoyed by the cortex. Sections should be preserved for microscopic examination. The extent of implication of the ganglia, pons, medulla, and spinal cord should also be examined.

Descending Sclerosis.—If again a greyish induration should be met with involving the white strands running down from the motor region of the brain to the internal capsule, or from the latter down into the crura, and on into the lateral columns of the cord, we are dealing with a secondary descending sclerosis (fasciculated sclerosis of Charcot).

B. *Colour.*—The alternating lighter and darker shadings of the cortical layers vary much in the depth of their hue, rendering the distinctness or differentiation of the individual layers more or less apparent. The variations in hue from the normal standard may be distributed in *mottled patches or laminated zones*, or *spread uniformly throughout the whole cortical envelope.* The student will do well to consider the conditions usually associated with alterations in the colour of the cortical layers, and first and most frequent amongst such conditions must be placed *alterations in the blood supply.* The next most frequent condition met with is *disintegration of nervous tissue;* and next to these in their relative order of occurrence we find *morbid deposits;* and lastly, *new growths* in the cortex.

Let us first consider the morbid conditions associated with unusual pallor.

a. Partial Mottling and Laminated Pallor.—A very slight acquaintance with the morbid appearances found in the brain of the insane will suffice to attract attention to the frequent presence of irregular, oval or circular patches of pallor, very limited in extent, and of a yellowish grey hue. These patches may extend throughout the whole depth of the grey matter or be limited to the superficial layers alone. Now this condition appears almost invariably attributable to anæmic zones, in which special systems of arterioles are involved. It may however be due to granular disintegration of nerve-cells and fatty accumulations in the perivascular sheaths.

A similar patchy pallor will also be frequently met with in the central ganglia, but here it will usually be found associated with fatty degeneration of the nuclei in the walls of the blood-vessels.

A portion of a convolution exhibiting this appearance should be placed on the freezing microtome, with the blotchy

aspect uppermost. On freezing it will be observed that the whole depth of cortex assumes a uniform pallor throughout, and sections cut off and floated in water *fail to exhibit the pale patches* if the latter be due to anæmic conditions simply; on the other hand, if due to structural disintegration, the appearance vanishes during the frozen state, only to return rapidly when the section is cut. The process of hardening by chrome salts dissipates all patches of pallor due to anæmic zones, for by both processes the blood vessels are more or less emptied of their contents, and a uniform tint results.

Apart from the presence of fatty or granular débris, the general pallor of the cortex may be due to great paucity of nerve-cells, whilst the blood supply being diminished the resulting pallor will be associated with a poor differentiation of layers. This apparent fusion of layers and loss of the sharp boundary lines seen in healthy brain is therefore a fact of some significance; it almost invariably points to general malnutrition and disintegration of nerve-cells. Pallor, however, is not necessarily associated with disintegration of nerve-cells, for the latter condition is frequently attended by vascular injection.

b. General Diffused Pallor.—I cannot do better here than describe to the student the appearances found in the case of a patient exhausted by phthisis, in which the typical *pallor* and *malnutrition* of the nervous centres is always so prominent a feature. Such cases are unfortunately so frequent in our asylums that the student will have little difficulty in procuring such a brain for study.

Upon removal of the extremely thin, blanched skull cap, and reflecting the dura mater, the subjacent membranes were seen separated widely from the surface of the brain, buoyed up by fluid which in part was clear and translucent, in part slightly turbid from films and flakes of lymph. The small arterial branches were not visible, the larger primary branches being alone apparent at the vertex. The venous system was represented by engorged trunks, their minutest radicles being well seen. The arachnoid where it crossed the sulci was more or less cloudy, the milky opalescence being due to interstitial change and lymph deposits. In the postero-parietal regions,

where the membranes were floated by the distending fluid off from the surface of the gyri, the smaller veins could be traced meandering through the fluid, and dipping down to enter the cortex. Tracing the course taken by these vessels, the smallest radicles were seen emerging from the cortex, uniting here and there with larger twigs, which ramify through the serous fluid and cross obliquely or directly over the summit of the convolution, to terminate in the primary veins which are formed by their convergence. These latter veins run up the sulci on either side of the convolution, exposed superficially and directed towards the median line or longitudinal fissure, where they terminate in the longitudinal sinus. To render their distribution more apparent, we have only to compress the orifice of one of the larger superficial veins near the median line, whilst with the handle of the scalpel placed on a distal part of the same vein, we include numerous converging radicles betwixt the occluded points. Upon gradually drawing the handle of the scalpel along the course of the vein upwards, the blood is projected into these radicles, and innumerable branchlets before invisible strike across the field over the convolution, or meander in tortuous arborescent forms through the subjacent fluid. The student will learn from the above examination the following facts:—

1st. The larger arteries being at the base, the smaller branches are alone seen near the vertex, and these usually deeply seated in the sulci.

2nd. All the larger blood-vessels usually exposed at the vertex are veins.

3rd. The direction taken by the minute venous radicles will be accurately learnt.

4th. A high degree of vascularity simulating passive congestion may be induced *artificially* with ease by slight force.

It is necessary to observe here that great caution should be observed by the student in hastily arriving at any conclusion as to the presence of congestion of the brain from a superficial view of the veins at the vertex—upon this point the tyro constantly errs. "We must make it a rule to consider hyperæmia of the cerebral membranes as proved only in those

cases where the finest vessels are also injected, and where the overloading of the cerebral vessels is not at all in proportion to the amount of blood in other organs."[1] So says Niemeyer, reiterating a caution constantly given by pathologists, and as frequently neglected. Proceeding with our study of the anæmic brain, the membranes were next stripped and two facts noted; they were slightly thicker and more tough than in health, and they were removed with extreme ease. The student must be familiar with the difficulty experienced in stripping the membranes from healthy brain, being recommended often to remove them under water. This difficulty is not alone due to the tenuity of the pia mater and arachnoid, but also to the fact that the former membrane is attached to the surface by the prolongations of connective cells recognised as Deiter's corpuscles. These are by no means numerous in health, yet sufficiently so to keep the pia mater in firm contact with the cortex. These cells are chiefly found in the neighbourhood of the vessels, where they enter the cortex from the pia mater, and both together form an impediment to easy stripping. In cases of subarachnoid serous effusion such as the one under consideration, the membranes have been floated up from the receding surface of the atrophic cortex, and the connections with Deiter's corpuscles have therefore been forced asunder. After noting fully the consistence of the brain according to the plan already given, a section was made exposing the centrum semi-ovale, and the following facts presented themselves.

1st. The blanched aspect of the cortex and the absence of any reddish striation of the upper layers such as is seen when the cortical vessels are full of blood. The hue of the cortex was *ashy grey throughout*, except in its most vascular zone, where perhaps the slightest warmth of tint was recognisable, but beyond this nowhere did any blush suggest the presence of its extremely elaborate vascular apparatus.

2nd. The layers were individually very poorly differentiated.

[1] Niemeyer's 'Practical Medicine,' translated by Drs. Humphrey and Hackley, vol. ii. p. 155.

3rd. The medulla was brilliant white, glistening, slightly reduced in consistence, and showed few or no puncta vasculosa.

The student will do well to impress the fact upon his mind that the vessels of the meninges may be tortuous and turgid with blood whilst the minute vascular supply of the cortex is absolutely diminished, and the layers appear blanched and anæmic. To a certain extent this was the case in the brain we have under consideration. How this occurs (passive or active hyperæmia of the membranes associated with anæmia of the cortex) will be explained in our next section, when speaking of hyperæmia of the brain. For the present, let the student mark well the fact that the *vascular condition of the membranes is no index*, as a rule, to the *state of the cerebral substance*.

The brilliant white glistening aspect of the medulla was due, as will be shown further on, to a slight degree of œdema of the brain substance.

Let us now place a small portion of a parietal convolution of thin and anæmic brain upon our freezing microtome and cut a fine section through the cortex—float it on the glass slide and drain off superfluous fluid. Take a hand lens and examine first by reflected light. The cortex is seen apparently to consist of—

1st. An outer light translucent zone, extending half way down.

2nd. A deep opaque white layer extending to the medulla, which is still more opaque and white in aspect.

Raise the slide and examine by transmitted light. We now see—

1st. The narrow translucent grey of the first or outer layer of the cortex.

2nd. A broad less translucent grey zone of the second and third layers.

3rd. A broad stripe of darker grey extending to the medulla, but separated at one-third its depth by a narrow blue belt.

We see thus by the naked eye the constitution of the cortex in this region of the brain, except that the narrow second layer cannot be defined from the third layer by *unaided vision*.

Let us turn to the cortex prior to section cutting and examine by means of a hand lens under reflected light, and the appearances are as follows:—

1st. A grey belt corresponding to the first layer.

2nd. A broad white belt divided midway by a narrow grey line.

3rd. A grey belt constituting the deepest layer.

We have, I think, gone sufficiently into the appearances presented by the anæmic brain for our present purpose. The examination of the various layers above recommended will prepare the student, not only for recognising the relative position of layers which have later on to be minutely studied, but will teach him to estimate roughly—

a. The differentiation of the layers by naked vision.

b. The relative depth of individual layers.

c. The atrophy or normal depth of the cortex.

d. The presence of morbid products.

Of the latter I need here only mention those cases of so-called miliary sclerosis in which the nodules can be distinctly seen in sections on examination by *reflected light* dotting the surface over with minute opalescent spots. For the present we must relegate such cases of morbid formations to the chapter on the microscopic appearance of the morbid brain, and now recapitulate shortly the points to be noted when examining by naked vision a cortex of unusual pallor:—

1st. The extent of pallor throughout the brain.

2nd. Its limitation in depth through the cortex.

3rd. Its disposition in patchy areas or otherwise.

4th. Its association with obscure laminar boundaries.

5th. Its association with pigmentary tints.

6th. Its association with altered consistence and œdema.

7th. Its reappearance in sections from frozen brain.

8th. Examination of sections by transmitted and reflected light.

Redness.—The depth of tint acquired by the grey layers of the cortex depends not alone on the presence of pigmented nerve-cells, but also on the far greater vascularity of the grey as compared with the white matter, whilst the layers most richly supplied with nerve elements possess also the more abundant

capillary supply. The more vascular the layer is, the deeper and warmer will be its tint. Now the capillaries of the cortex are of remarkably fine calibre, and hence, when the ultimate arterioles are injected, the appearance resulting is that of a *uniform rosy blush* more or less dark, the surface *smooth* and *swollen*. This uniform coloration, if at all extreme, is often attended by slight extravasations of blood, and the tissue around presents a diffused staining from blood pigment. Such are the conditions found in genuine congestion of the cerebral substance, and the analogous condition is readily recognisable in the soft membranes. The student must make himself thoroughly acquainted with the evidences of a genuine active or passive congestion of the brain, and learn to distinguish it from actual inflammation where, as already pointed out, the *consistence* as well as *colour* are profoundly implicated and the *specific gravity also affected.*

a. Congested zones.—One of the most frequent appearances in the cortex of the acute forms of insanity is a bright arterial zone which bounds the confines of the white and grey matter of the convolutions, following out accurately the direction of the innermost cortical layer. This congested belt corresponds to the horizontally disposed nexus of blood-vessels into which the larger straight cortical arteries empty themselves after passing through the various cortical layers. Any undue engorgement of the vessels of the pia mater will necessarily affect these larger blood-vessels, and from their general arrangement a mechanical element is introduced which is significant of a safety-valve action for the cortex in cases of vascular engorgement, relieving the cortex somewhat by their distension from congestive conditions. It is on this account, I believe, that this linear vascularity is so often apparent without a corresponding blush in the cortical layers, indicative of distension of the minute capillaries.

b. Limited Patchy Congestion of Cortex.—When, however, the cortex is itself congested the student will frequently observe irregular bright red patches, suggestive, like the anæmic patches already referred to, of the implication of minute vascular areas, whilst leading down to them are seen the distended straight cortical vessels, giving the upper layers

a reddish streaked aspect. The last act of arterial contraction in which the smaller arterioles have failed to empty themselves into the venous system may in part explain this appearance, and caution is necessary lest we hastily assume that this state is the result of morbid activity. The student should therefore examine closely the injected part by the microscope and look for evidence of minute extravasations, hæmatine crystals, and staining as well as broken down texture, or other results of inflammatory action and of congestion. This blotchy red aspect of the cortex reappears very frequently in the medulla in similar cases, and is indicative of engorgement of the minute capillaries of these regions.

c. Puncta Vasculosa a fallacious guide.—It is customary, in judging of the degree of engorgement of the vessels of the white matter, to be guided by the paucity or abundance of the puncta vasculosa caused by section of the engorged channels; in other words, the number of drops of blood oozing from the divided vessels are supposed to form some criterion of the engorged condition of the vascular system here. It has been shown by Niemeyer that this is no safe criterion, as

1st. The number of puncta vasculosa vary greatly with the fluidity of the blood, which is an important element in their causation.

2nd. They are often almost entirely absent, when we have had undisputed evidence of intense vascular engorgement during life.

The student must therefore be on his guard against attributing too much importance to abundant bloody points on section of the medulla, whilst he should never fail to note whether this condition is or is not associated with distension of the minute capillaries, as indicated by fine diffuse patchy redness.

I need not here dwell upon the changes of colour due to inflammatory action, as these have been already disposed of when illustrating anomalies of consistence; but I must here repeat the important caution, that the student be not misled by expecting to find engorgement and redness in all cases of encephalitis. The larger number of such cases present no undue red coloration.

Results of Intra-cranial Pressure.—The student must invariably allow for conditions of increased intra-cranial (*but extra-vascular*) pressure. When the cranial cavity is encroached upon by a tumour, abscess, hæmorrhage or serous accumulation, compensation is made by the outflow of the subarachnoid fluid into the spinal meninges. The *limit of this compensatory arrangement* is soon reached, and then the only available space is that gained at the expense of the *general vascular calibre.* Encroachments upon the cranial cavity therefore eventually empty the blood-vessels, causing partial or more or less general anæmia prior to compression of the brain substance itself. It is therefore far from improbable that, as Niemeyer teaches, engorgement of the blood-vessels may reach such a degree that after the limit of subarachnoid compensation has been reached, effusion of serum occurs into the perivascular spaces and brain tissue, and suffices to compress the minute blood-vessels and capillaries. Hence we may find, in such cases, an association of marked cerebral anæmia with tortuous and engorged blood-vessels in the meninges.

Results of Intra-thoracic Pressure.—Venous engorgement of the brain is a frequent result of obstructed circulation through the medium of pleuritic effusions, intra-thoracic growths, fibroid induration and other changes obstructing or obliterating the vascular system of the lungs. Occasionally, though rarely, there is found an extraordinary engorgement and varicosity of the veins of the pia mater—the vessels winding in all directions and, as Rokitansky states,[1] even in spirally twisted coils and intestine-like circumvolutions. In these cases the brain substance will be found dark and engorged, full of puncta vasculosa and frequent miliary apoplexies. In one very typical case, occurring at West Riding Asylum, the brain surface was actually concealed over extensive tracts by a vast development of varices and contorted vessels, and when cut into, the grey and white substance was not only deeply engorged but presented dozens of miliary hæmorrhages forming patches varying from a pea to a florin in extent, and in all

[1] 'Pathological Anatomy,' Sydenham Soc., vol. iv. pp. 372-3.

degrees from the purely punctiform to the diffuse uniform extravasation. The appearances of a section of this brain are well shown in a preparation at the Asylum Museum. A similar case was exhibited some years back by Dr. Coupland at the Pathological Society of London.[1] In his case the mechanical hyperæmia was due to acute bronchitis; in the Asylum case, the immediate cause was the supervention of capillary bronchitis upon fibroid lungs in a patient who gave a history of intemperance. Its association with drink has been recognised by Rokitansky. The occurrence, then, of such cases should direct the student's attention to the effects of intra-thoracic pressure upon the venous circulation of the brain.

Volumetric Methods.

Cubic Measurement of Brain.—The bulk or volume of the encephalon can be very readily ascertained, and, considering the important information it affords, it is a process too frequently neglected in our post-mortem rooms. The volume of the brain is estimated by its displacement of fluid. Dr. Hack Tuke has detailed, in the January number of the 'British and Foreign Medical Review' for 1855, the result of examination by this method of sixty-three brains, together with the capacity of the crania. The vessel used by this observer was one of convenient size and shape, with a capacious spout placed at an acute angle with the sides. Water is poured into this vessel up to the level of the spout. Fluid contained within the ventricles and subarachnoid space is allowed first to escape by several long incisions, and then the brain, including the medulla oblongata, is immersed—the displaced water, as it escapes from the spout, being caught and measured, affording an exact criterion of the actual bulk of the brain. The student may, however, prefer the use of a graduated vessel upon which he can at once read off the displacement of fluid, but the exact graduation of large vessels such as would be required is open to several fallacies; and these possible sources of error are, of course, wholly avoided by Dr. Hack Tuke's method. If such a vessel as that used by Dr. Tuke be not at

[1] Reported in the 'Lancet' for January 11, 1879.

hand, a ready method is the one I have used, as follows:—half fill with water an inverted bell glass of sufficient dimensions, and mark the level of the fluid. Now immerse the brain in the fluid, and note the level of displacement, after which the brain is removed; if the original level is not exactly maintained owing to imbibition by the brain, pour in sufficient fluid to compensate for the loss. Water is now poured in from a graduated measure up to the displacement level, the amount required for this purpose giving us the volume of the brain. The volume should be estimated in cubic centimeters and cubic inches.

When describing the methods of section-cutting I shall refer to a vessel which will answer well for the volumetric estimate of the brain. It consists simply of a large glass vessel such as is used for the preservation of brain in museums, but fitted with a stop-cock arrangement in the side near the bottom of the vessel. It is filled with sufficient water to cover the brain, the level being marked or indicated by a weighted float. The brain is next immersed, and whilst so immersed, the fluid of displacement is run off by the stop-cock, and measured in a graduated vessel. Again, in lieu of a graduated vessel, the student may employ a float, moveable along a graduated weighted stem (graduated, of course, for the vessel used). Such a float should terminate below in a brass button, which rests against the bottom of the vessel, keeping the graduated stem in a vertical position, whilst the float freely moves with the rising or falling level of the fluid along the graduated stem.

When referring to the specific gravity of the brain, an apparatus will be described whereby volume as well as weight of brain are readily measured. Such an apparatus has the recommendation of being cheap and efficient. Whatever arrangement be adopted, we should not rest content with the estimate of the volume of the encephalon alone. Each hemisphere should be separately measured, as also the cerebellum, pons, and medulla. It will be found convenient to use smaller and more delicately graduated vessels for the latter.

Cranial Capacity.—This should be estimated with a view to

a comparison between the actual volume and weight of the brain and the dimensions of the cranial cavity, as well as for comparison with the capacity of average-sized skulls. The relationship between cranial capacity and brain weight has been shewn by Dr. Barnard Davis, who has adopted the general rule that a deduction of about fifteen per cent. from the capacity of the cranium gives the "capacity" of the brain, whereby its weight may be readily calculated.[1] The large bulk of cranio-metric observations having been taken upon the dried skull, we should when dealing with the fresh subject make allowances for slight divergence; and, in order to approximate the conditions, the dura mater should be wholly removed from the skull cap and the base. When, however, our intention is to estimate the difference in any single case between cranial capacity and brain-volume, rather than for more general comparative purposes, it will be necessary to open the skull prior to opening the thorax so as to avoid emptying the venous sinuses, whilst at the same time the dura mater must not be stripped away from the skull cap nor the base be uncovered. With the object of estimating the amount of cerebral atrophy, a valuable series of observations were made by Dr. Hack Tuke, and the process he adopted is described as follows:— "The foramina at the base of the brain are carefully plugged with tenacious clay—that used by statuaries for modelling answers best; a small triangular piece of the frontal bone is removed with the saw; the calvarium is re-adjusted to the base, the dura mater being left attached. The space left by the attrition of the saw in removing the calvarium is filled with clay; and a narrow bandage with clay spread upon it, is made to surround the cranium three or four times, covering this space. If this manipulation has been carefully done, the cavity of the cranium will now be found as tight as a bottle. Sixty fluid ounces of water having been measured, a sufficient quantity to fill the cranial cavity is now poured into it by means of a funnel, through the orifice in the frontal bone, taking care that the stream does not wash away the luting of the foramina. The fluid which remains, after having filled

[1] On the weight of the brain in the different races of Man. Philos. Trans., 1868, pp. 506 and 526.

the cranial cavity is measured, and being deducted from the sixty ounces gives the amount employed. To this must be added half an ounce for the space occupied by the luting." Having thus obtained the cranial capacity, he deducts from it the brain volume, and obtains thus the exact measurement of shrinking or atrophy. Millet seed and sand have been used for measuring the capacity of the cranial cavity, and the latter in the dried skull has answered admirably. It must, however, be borne in mind that a fallacy may be introduced by the employment for this purpose of materials liable to be influenced by temperature. Fluids are, of course, more open to this objection, their expansion by heat being proportionately more rapid than solids, and hence it would appear that sand is preferable to water in these investigations. Mustard seed was employed by Professor Flower in his extensive series of researches.[1] The method adopted by myself arose from my employment of solid paraffin in obtaining casts of the brain surface, and the interior of the skull. It was soon apparent that the finest and most delicate impressions and most perfect casts could be obtained by the use of this substance, and that it did not share in the great disadvantage which accrues from the use of plaster of Paris, viz. that of great contraction during solidification. It is in this respect also far superior to white wax, which, as is well known, contracts much whilst solidifying. I proceed as follows:—Fill up the foramina at the base as previously described. A triangular or wedge-shaped piece is now sawn out of the occipital bone after removal of the calvarium, but retained in situ. The base is now filled up by melted paraffin, the skull cap replaced and fastened by luting, just as in Dr. Tuke's process, having previously trephined a piece out of the frontal bone. Through the latter orifice more of the paraffin is poured in until the cranial cavity is filled. When cool and solid remove the calvarium, as well as the wedge-shaped piece of bone from the occiput, and then gentle pressure from behind tilts the solid mass out of the cranium, when it will be found to form an exquisite mould of the interior. The mould thus obtained is

[1] The capacity of the crania contained in the Hunterian Museum of the Royal College of Surgeons was obtained by Professor Flower in this manner.

now to be measured by displacement, whence we obtain the cranial capacity. This method has afforded me great satisfaction, as it gives data of immense value at the expense of very little trouble: it supplies us with a method of—

1st. Estimating cranial capacity.

2nd. Gives us an exact mould of the cranial cavity.

3rd. As a permanent record—numerous linear angular measurement, and volume measurements may be obtained.

CRANIAL CAPACITY IN DIFFERENT HUMAN RACES.

CRANIAL CAPACITY.	Modern Parisians.	Parisians of 12th Cent.	Ancient Egyptians.	Negroes.	Australians.
Cubic Centimeters.					
1200 to 1300 . .	0·0	0·0	0·0	7·4	45·0
1300 to 1400 . .	10·4	7·5	12·1	35·2	25·0
1400 to 1500 . .	14·3	37 3	42·5	33·4	20·0
1500 to 1600 . .	46·7	29·8	36·4	14·7	10·0
1600 to 1700 . .	16·9	20 9	9·0	9·3	0·0
1700 to 1800 . .	6·5	4·5	0·0	0·0	0·0
1800 to 1900 . .	5·2	0·0	0·0	0·0	0·0

From some invaluable measurements by the late lamented Broca,[1] it was found that of 115 skulls of individuals living in the twelfth century, the *average* capacity was 1425·98 cubic centimeters; whilst of 125 skulls of the nineteenth century the average capacity was 1461·53 cubic centimeters. The accompanying table of percentage will be found of use by those interested in the subject of cranial capacity. It is given by Le Bon as illustrative of the relationship of race to cranial capacity.

[1] Quoted by Nogt, "Lectures on Man." (Anthrop. Soc.)

(To be continued.)

Critical Digests and Notices of Books.

On the Circulation of the Blood in the Human Brain. By Professor Mosso. Turin.

(*Sulla Circolazione del Sangue nel Cervello dell' Uomo.* Roma, 1880. 4to, pp. 127.)

The observations on which this important memoir is founded were made on three different individuals, two of them still surviving, whose histories are shortly the following:—

1. Catherine X——, a countrywoman, æt. 37, suffered in 1866 from syphilitic sores, which were followed by four successive attacks of secondary and tertiary syphilis, ending in 1875 in the destruction of nearly all the frontal bone, and a great portion of the two parietals. The removal of this large amount of bone left a large opening in the head, through which the pulsations of the vessels of the brain could be observed.

2. Giovanni Thron, an epileptic boy of 11 years of age, met with an accident at the age of 18 months, which destroyed a large part of the cranial substance in the right temporo-parietal region. So far as could be made out from the scanty history, it was not till eighteen months had passed that any important brain symptoms appeared; but at 3 years of age he began to suffer from epileptiform and epileptic seizures. When this boy came under Dr. Mosso's care in 1877, the elliptical opening in the skull measured 70 mm. by 35. On pressing firmly with the finger no pulsations could be felt through the hard membrane which covered the opening. The boy died, in Dr. Mosso's absence, of an acute gastro-intestinal catarrh, and at the post-mortem examination it was found that the cerebral lesion involved the posterior extremity

of the horizontal ramus of the Silvian fissure, the anterior part of the inferior parietal convolution (*lobulus supra-marginalis*), and ended in the middle part of the ascending parietal convolution. Inferiorly it extended as far as the three temporo-sphenoidal convolutions to a part of the angular gyrus. No motor paralysis was discovered during life.

3. Michele Bertino, æt. 37, in July 1877 was struck on the head by a brick weighing 3 kilogrammes, which fell from the hand of a workman who, at the height of 14 metres, was employed on the roof of the campanile of his native village. The lacerated contusion caused by this accident was irregularly triangular in shape, 7 centimetres by 4, and corresponded with the frontal region, of which the bone sustained a compound comminuted fracture. He was treated by a surgeon, who removed portions of brick and of bone from the wound, and who says that Bertino was quite conscious, and could speak when he saw him laid on a bed immediately after the accident; but Bertino himself, in his account of the occurrence, told Dr. Mosso that he remembered nothing, not even having received the blow, and that he believes he recovered consciousness about an hour after. After remaining twenty-four days in bed Bertino went to a neighbouring hospital to see another doctor, and in eight weeks from the occurrence of the accident he went to Turin. There was then a circular opening in the skull, of a diameter of 25 mm., in close proximity to the fronto-parietal suture. The general appearance of the opening, looked at from above downwards, was funnel-shaped, and the floor was formed by a red membrane with some granulations. When the patient stood erect, or sat, pulsations could be observed in the cavity synchronous with the radial pulse. There was an abundant secretion of pus from the cavity, whose capacity, when the patient was at rest, was about 5 cubic centimetres of water. In Bertino's account to Dr. Mosso he said that he found himself able to follow his occupation as usual about a month after he had left the hospital. The only difference he observed was, that when he put forth a special effort or stooped, he had a sensation as if his brain went upwards. In 1878 he had a fainting-fit; but in other respects he has remained quite well and like his former self, except

that he has become more timid and less courageous than before. The apparatus employed to measure the cerebral and brachial pulse consisted of various ingenious modifications of Marey's conjugate tympana, with special adaptations of gutta-percha plates, &c., as also of the cardiograph, sphygmograph, hydrosphygmograph, and plethosphygmograph—the last instrument, as the name implies, being used to measure the volume of the pulse.

As to the form of the pulse-curve, Professor Mosso says that the *tricuspid* pulse-tracing characteristic of the circulation in the brain can be produced in the radial and carotid arteries, and in the nasal cavity at will. This tricuspid tracing is so called because it shows three elevations, of which the central one is the highest, and the other two arise directly from the sides. After Landois, he distinguishes three parts in the tracing, the *ascending*, the *vertex*, and the *descending* part, and, after him also, elevations observed in the ascending part are called *anacrotic*, while elevations observed in the descending part are called *catacrotic*.

Dr. Mosso goes on to show that the tricuspid tracing depends on the state of the vessels. In fact, when a contraction of their walls is produced, an anacrotic pulse becomes a catacrotic. Figs. 6 and 8 show a tricuspid pulse in the forearm becoming catacrotic by cerebral activity. In Fig. 9 it is shown that when the "secondary elevation" (S) is non-existent in the radial pulse-tracing, it can be made to appear by such a contraction of the vessels as is induced by cerebral activity.

Fig. 7 shows how a tricuspid radial pulse can be converted into a catacrotic by inhalation of ammonia, at the same time that, as shown by the plethosphygmograph, the volume of the pulse was diminished by 16 cubic centimetres, and even by as much as 20 cubic centimetres. Figs. 10, 11, 12 deal with the changes in the pulse-tracing observed during fasting, and after eating. The general result is, that the absorption of food induces a catacrotic pulse-tracing, with a secondary elevation, as well in the radial vessel as in the brain itself.

Dr. Mosso brings forward the following considerations to prove that the tricuspid pulse is a phenomenon which depends on the state of the vessels. 1. The cerebral pulse-

tracing is often tricuspid at the same time that the carotid is catacrotic. 2. One radial tracing may be catacrotic, while the other is tricuspid. 3. A catacrotic radial pulse can be rendered tricuspid by heat, by cold, by muscular contraction stimulated by an induced current, and by voluntary muscular contraction, &c. Experiments are described to prove in detail these special points.

In Chapter IV. Dr. Mosso deals with the interesting question, "What are the effects on the pulse-tracing, of Thought and Emotion?" Fig. 21 shows that there was a large augmentation in the size of the cerebral tracings. 1. When mental activity was excited in Bertino by the question, how many eggs make twelve dozen? 2. By a reproof from Dr. Mosso to Bertino for having involuntarily moved his finger, so spoiling the radial tracing which was in the act of being compared with the cerebral. 3. By the clock striking when Bertino did not expect it; and 4. When Dr. Mosso asked him whether he was accustomed to say the *Ave Maria* at mid-day.

Dr. Mosso next proceeds to meet the objection raised by Professor Franck, that changes in the circulation might depend on changes in the respiratory movements. The author denies this for the following reasons:—

1*st*. Because often the forearm tracing shows diminution of volume during cerebral activity, while the respiratory movements remain unaltered in rhythm and depth.

2*nd*. Because we often find that the volume of the cerebral circulation increases during cerebral activity, while the respiration remains unaffected.

3*rd*. Because the characters of the sphygmic curve of the forearm show that a real contraction of the vessels takes place, which does not correspond with what is observed when a simple modification of respiration occurs; and

4*th*. Because we do not observe similar changes in the pulse of the brain and the forearm, which we should do if they depended on changes of the respiration, since respiration bears the same relation to the cerebral- that it does to the forearm-circulation.

Section 4 of this interesting chapter deals with examples of the law which Dr. Mosso believes he has established, that "the

emotion which is produced when passing from profound rest to a state of cerebral activity is always accompanied by a modification of pulse."

In Chapter V. the author deals with "Sleep in its Relations to the Circulation in the Brain." The author had formerly shown that in passing from the waking state into sleep there occurs a dilatation and relaxation of the vessels of the forearm, and that the act of waking is always accompanied by a contraction of the vessels. He has made experiments on natural sleep and artificial (induced by chloral hydrate) in the cases of Catherine X—— of Thron, of Signor Caudana, and of Bertino. The general result (to which there are, however, a few exceptions) is, that in the act of waking there occurs an augmentation in the size of the pulse-tracings. So much is this the case that frequently there is observed a similar augmentation, even when an unsuccessful attempt is made to wake the subject of experiment, such, for example, as calling him by name, but not so loud as to wake him. Dr. Mosso draws Fig. 28 to show that even the slight noise caused by his rising on his feet to call Bertino quietly (though the calling was not loud enough to wake him) sufficed to elevate the pulse-tracings a little. The tracing, besides, which was catacrotic before Bertino was called, became anacrotic afterwards, and remained so for some time. There were occasional augmentations in the size of the pulse-tracings which could not be explained (Table VI., line 26).

From Fig. 29, and also from several of the lines of Table VII., it appears that sometimes in the act of waking, the brain may undergo a diminution of volume, and may contain, in fact, less blood than during sleep, which, says Dr. Mosso, "suffices to render untenable the hypothesis which attributes sleep to a state of anæmia of the brain."

Chapter VI. deals with considerations regarding the nature of sleep, and of the phenomena which accompany it.

A modification of the circulation, it has been shown, takes place in passing from waking to profound rest, and from the latter to sleep. In sleep there occurs a dilatation of the vessels of the extremities which can be measured in the forearm by the plethosphygmograph. It corresponds with a relaxation

of the vascular walls. Every excitation from without causes a contraction of the vessels of the forearm and a subsequent increase of the blood-pressure, causing a larger flow of blood to the brain. On contraction of the vessels of the brain, which takes place on sudden waking, the general increase of pressure produces similarly a greater velocity of blood in the cerebral hemispheres. In sleep the heart movements become somewhat slower, and the changes above-mentioned, produced in the organism during waking hours by cerebral activity, are reproduced in sleep by external actions insufficient to wake the sleeper. Thus a sound, a voice, a touch, may modify the rhythm of the respiration, contract the vessels of the forearm, increase the pressure and flow of blood to the brain, and accelerate the rhythm of the heart; and yet the sleeper, on being wakened, asserts that he has no consciousness of having been affected by these causes. Dr. Mosso has called such influences, not depending on exaggerated respiration, and not leaving any trace in consciousness, *spontaneous*, and quotations are made from Carpenter and Maudesley in support of his view of them.

Just as was the case with the vascular supply to the brain, a noise, insufficient to wake the sleeper, may modify both the thoracic and abdominal respiration. Thus, in such a case the expiratory curve, which is a little delayed in time, is considerably increased in size when it does appear. After this there occurs a deeper and quicker inspiration; after which there takes place a slight commencement of expiration, and then a more marked and stronger expiration. On comparing the abdominal with the thoracic respiration, a want of correspondence between them is observed. When the thorax is making the expiratory movement the abdomen remains fixed; and when the thoracic inspiration begins, then takes place on the contrary an expiratory movement in the abdomen. But, during the continuance of the thoracic inspiration, elevation of the abdomen also occurs corresponding with inspiration. The cause of this is that, whereas during waking hours the diaphragm is one of the most active of the muscles, in sleep it becomes inactive and almost paralysed. During waking the abdomen rises with the elevation of the thorax, because the diaphragm falls synchronously with the movements of the

respiratory muscles. But when in sleep the diaphragm ceases to contract, dilatation of the thorax is accompanied by sinking of the abdomen, because the diaphragm is drawn up in inspiration like an inert membrane. When the sleeper wakes, the normal relations are speedily re-established.

The unconscious changes which occur during sleep are very remarkable, and, in the opinion of Dr. Mosso, with which Herbert Spencer's view mainly coincides, serve the purpose of increasing the blood-supply to the brain, and so of waking the animal when in danger. The animal which can bring most energy to bear in a short time has the best chance in the struggle for existence, since it may overcome an animal stronger than itself on the average, but which cannot put forth so much energy at once. In order to repair waste, and induce a high state of activity, animals must sleep profoundly; but during deep sleep a voice, a distant noise, a ray of light falling on the eyelids, or a slight touch, excites the respiration, contracts the vessels of the extremities, increases the energy of the heart's action, and the frequency of its beats, increases the blood pressure, and drives more blood to the brain; and thus the animal can pass from profound repose into complete activity in a time so short as to enable it to escape danger, which would overwhelm one whose processes were slower or less well adapted for their purpose.

In Chapter VII. is considered the occurrence of certain undulations in the pulse-tracings not dependent either on the respiratory movements (those so produced being termed *oscillations*), nor on the cardiac systole (called *pulsations*). These undulations are termed *spontaneous*, not in the strict sense of the word, of course, but in order to indicate that the cause of the phenomenon is not quite known. Dr. Mosso says they are due to changes in the elasticity of the vessels, and they are not the same as those which may be called *passive*, and which depend on an increase or diminution of the blood pressure without the vessels of the organ itself taking any active part in the formation. He concludes that the spontaneous undulations of the vessels constitute a phenomenon which has no relation, either with the innervation, or with the movements of the respiratory apparatus (Traube and Hering); that it is

produced without its being possible for us to believe that it depends on a certain venosity of the arterial blood (Cyon, Hering); on the accumulation of CO_2, or on the deficiency of O. The spontaneous undulations of the cerebral vessels have no constant rhythm, and the cerebral tracing may often remain for a long time horizontal with no other elevations than those of the pulse; and this even when, in the arm, respiratory oscillations are perceived.[1] The remaining sections of this chapter are occupied with delineations of the undulations in the tracings caused by electric irritation of the vagus in dogs, of those caused by slowing of the heart's action, and of the periodic variations occurring naturally in the carotid pulse, and of those induced by the action of chloroform.

In Chapter VIII. Dr. Mosso details some observations on vascular movements made on the ears of rabbits. He thought that previous observations might be vitiated by the emotion of the animals, and therefore constructed a cage with holes so placed as to enable him to watch the animals, without being himself observed. The results were quite in accordance with his expectation. He found that the contractions and dilatations which were described as taking place in the rabbit's ear, and which were ascribed to what Dr. Schiff called *the accessory heart*, ceased almost entirely when the animal was in complete repose. In profound tranquillity the artery of the ear would remain dilated sometimes for hours together. On the other hand, a whistle, a cry, the sound of the bells of the neighbouring church, the bark of a dog, and even a ray of light falling on the cage, all induced contraction of the vessels. His conclusion is that "the movements of the blood-vessels in the ear of the rabbit are related to the emotions and to the psychic state of the animal."

In Chapter IX. the influence of the respiratory movements upon the circulation of the brain and lungs is discussed. Speaking generally, it may be said that regular and easy

[1] May not these undulations depend, like so many other organic phenomena, on the fact that there is no stillness or sameness in life; so that life would be represented diagrammatically, not by a straight line, but by a wave? And may not the cause be that the cell, the ultimate element of organic structure, is itself in a continual swing of movement, now swelling and now shrinking, but never at absolute rest?—Tr.

respiration has so small an influence on the movements of the brain that it almost escapes observation. But careful observation discovers that in inspiration there is a rather lower pulse-tracing, and that in expiration the elevation of the pulse-tracings is increased. Not only is this so; but the elevation of the pulse-tracing is more or less proportionate to the depth of the inspiration and expiration, being higher than natural in the full expiration which succeeds a deep inspiration. In Fig. 49 the author shows an elevation produced in the cerebral pulse-tracings, 1st by a mental act (propounding to the subject of experiment the question how many are 6×45); and 2nd, by an involuntary deep inspiration. There is a difference between the elevations in these two sets of circumstances, however, which consists in this—that in the former the brain is increased in volume, while the circulation of the forearm is diminished; while in expiration both the volume of the brain and that of the forearm are increased, though the increase is proportionately greater in the brain than in the forearm, due probably to its richer blood supply.

As to the mechanical causes affecting the circulation during inspiration and expiration, little remains to be added to Haller's statement that "in inspiration the brain diminishes in volume because the blood recedes from the great vessels of the brain, but that in expiration it swells and increases in size because the blood is retained." Dr. Mosso details experiments which show that the jugular pulse (in a previous work shown to be the *negative* of the arterial pulse) disappears when the breath is held; and that the respiratory oscillations visible in the tracing of the forearm disappear when a pressure of 20 centimetres is applied to the surface of the whole arm.

In the lower limbs, on the contrary, there is an increase in volume during inspiration, and a diminution in the beginning of expiration. This is the case both in normal inspiration and in slower and deeper breathing. It is noticeable that a rapid diminution in volume takes place in the beginning of expiration.

The brain of Bertino was found to diminish rapidly in volume during a profound rapid inspiration; the systoles of the heart became more frequent, and the cerebral pulse

nearly disappeared. In the next inspiration the brain rapidly swelled; the systoles became less frequent, and the pulse very strong. After the expiration was finished a second diminution occurred in the volume of the brain, and a very small pulse was observed.

In this chapter Dr. Mosso describes a set of ingenious experiments made to determine the quantities of blood going to and coming from the lungs respectively, and the quantity of residual blood remaining. From these experiments, which our limits do not permit of our detailing, it appears that in inspiration more blood enters the lungs than issues from them; and in expiration the efflux is greater than the influx. A residuum of 15 or 16 cubic centimetres of blood was left in the lungs, and it was found that the blood did not issue from the lungs so rapidly as it entered.

Chapter X. deals with criticisms of various researches on the circulation in the lungs.

In his attempts to determine in Chapter XI. the influence of the respiratory movements upon the general pressure of the blood, the author agrees with Einbrodt and Ludwig "that the pressure obtains an increase during inspiration, inasmuch as each cardiac systole attains a tension greater than the preceding pulsation." When the blood-pressure was diminished by hæmorrhage, it was found that in inspiration the pressure descended; in expiration it was first raised and then descended. This was the opposite condition to the former. Comparing the state of fasting with that existing after a meal, it was found that after eating, the oscillations of the forearm pulse were more marked than in the fasting state, although the respiratory movements were less deep and full.

After inhalation of nitrite of amyl the elevations of the pulse-tracings of the brain are much increased, and the dicrotism is much more marked. In Bertino the carotid tracing, which normally consists of three elevations, of which the two last occur almost at the same level, tended under the influence of nitrite of amyl to eliminate the first of the two, while the second descended and became more pronounced.

The chapter on cerebral hyperæmia and anæmia is very interesting. Dr. Mosso says: "Were I asked which of all the

functions of organs is most dependent on even the slightest change which takes place in the material interchange, I should without hesitation reply, that consciousness is that function." The following experiment shows the prodigious activity of material interchange in the brain. On the 29th of September, at 1 o'clock, Dr. Mosso and Dr. Paoli made some observations on cerebral anæmia in the case of Bertino, by means of the gutta-percha plate and the hydrosphygmograph. Comparative tracings of the brain and arm were taken. Dr. Paoli applied pressure with his fingers on both carotids. The first two elevations were higher than before, but the third was much smaller, and the brain rapidly diminished in volume. After the 8th cardiac systole the pulse slowed notably, and in the brain was so small as to be scarcely perceptible. At the 12th pulsation, that is after about eight seconds of cerebral anæmia, Bertino was seized with an accession of convulsions; the face pale and eyes turned upward. Dr. Paoli immediately set free the carotids. As soon as Dr. Mosso saw that Bertino opened his eyes as if in wonder at finding himself in that place in that position, he tried without delay to continue the registration of the pulse: but the brain had undergone such an increase in volume, and the arm had moved so much that it was twenty seconds after the first onset of the convulsions before he could take up again the registration of the cerebral pulse, and a minute more before the forearm pulse could be registered. Seeing that there was nothing noticeable beyond a strong increase of the cerebral pulse, he suspended the observation.

Bertino asked them to proceed with their observations; but they thought it better not to do so.

Dr. Mosso comments on the remarkable dependence of the psychical functions of the brain on the interchange of its component parts. No other parts of the body manifest this to the same extent, not even the sense organs. We can produce an ischæmia of the whole arm, and literally expel the blood by an elastic bandage for twenty or even twenty-five minutes, without depriving the sensory apparatus of the nervous system of its power to transmit tactile calorific or painful impressions; while in the brain a diminution, not to speak of an arrest, of

the flow of blood for a space of time 300 times less is sufficient to abolish consciousness.

Some observations on the circulation of the blood in the sound cranium terminate this valuable memoir.

A. Rabagliati.

Des Contre-Indications à l'Anesthésie Chirurgicale. Par le Dr. H. Duret. 8vo., pp. 278. Paris, 1880.

In this highly able and instructive memoir M. Duret discusses the physiological action of anæsthetics on man and the lower animals, the causes of death as determined by physiological experiment, and by a review of all the reported cases of death from chloroform in man; the general and special contra-indications of anæsthesia in surgical practice; and the relative advantages and disadvantages of different anæsthetics. The work is so full of detail as not to admit of analysis within reasonable limits, and we would refer all who are interested in the important questions discussed to the original memoir itself. We would only briefly indicate M. Duret's generalisations as to the mode of action of anæsthetics. Anæsthetics may be administered in the great majority of cases with perfect safety. They cause temporary annihilation of the functions of the cerebral hemispheres and spinal cord, characterised by sleep, anæsthesia and muscular relaxation. The medulla oblongata alone continues its activity, and presides over the vital functions of circulation and respiration. During the whole period of narcosis, the activity of the medulla oblongata must be specially guarded. As a rule there are premonitory indications of the failure of this centre which must not be disregarded. But the medulla is liable to successive shocks which menace its vitality, and fatal results may ensue, though comparatively rarely, at various stages of the narcosis.

1. At the commencement of the inhalation, by reflex shock due to irritation of the respiratory passages.

2. At a later stage, before narcosis is complete, by bulbar shock, induced by the first waves of the anæsthetic vapours reaching the medullary centres and causing syncope and asphyxia.

3. In the later stages death supervenes by paralysis of the respiratory and cardiac centres. Usually the respiratory centres fail first, and asphyxia precedes syncope. But in certain morbid conditions the heart is the first to cease.

4. Lastly, death may be caused during the narcosis by traumatic shock, acting on centres already depressed and enfeebled.

After the narcosis death may result from general exhaustion of the nerve centres.

D. Ferrier.

Westphal on the Knee-Phenomenon. Berl. Klin. Wochenschr. 1881, No. 1.

As a contribution in answer to the question whether an anatomical lesion of the posterior root-zones is already present at a time when abolition of the knee-phenomenon is the only sign of the presence of tabes, Prof. Westphal relates a case of which the following are the chief points—Neuropathic inheritance; complete amaurosis three years before death; mental aberration requiring restraint at about the same period; trifling difficulty of articulation; knee-phenomenon found normal at various times during the progress of the case; no tabetic signs; three months before death, abolition of the knee-phenomenon on both sides; no other tabetic signs; death by intestinal complication. P.M.—Lesion limited to posterior root-zones, and posterior part of lateral regions; posterior roots and columns of Goll normal.

The author links the knee-sign with the lesion of the post. root-zones, not with that in the lateral columns, because on the one hand he has found after tabes with absent knee-phenomenon, lesion of the posterior root-zones, without lesion in the lateral columns, on the other hand he has found the lateral lesion in cases where the knee-phenomenon had been normal or increased.

Referring to the supposed uncertainty of the well-known tabetic test, he states that the cases called tabes, where knee- and foot-phenomenon were present, have been miscalled tabes, though there may have been ataxic symptoms present; he

says that such cases are presumably cases of insular sclerosis, and emphasises the statement that in no case of well-authenticated tabes is the knee-phenomenon present. With regard to its absence in normal persons, he states that he has never failed to find it present, even in cases of its pretended absence; still he admits that it may be absent if from any cause the tendon is not susceptible of the sudden extension, as from its shortness in thick-legged persons, or the presence of much fat beneath it. Further, the test may fail from excess or deficiency of the muscular tonus, which he regards as its principal condition. And finally, if the reaction were absent in an apparently normal person, he should think that that person is not normal, but the subject of latent spinal mischief; if, the reaction having been absent, it should reappear, he would be inclined to diagnose retrogression of incipient mischief, and to consider the fact as a hopeful feature in the history of a condition usually regarded as fatally progressive.

In conclusion, Prof. Westphal signifies his maintenance of his original assertion that the knee-phenomenon is not a simple reflex, but a complex manifestation, intimately related to a muscular tonus, which is possibly reflex. He considers that lesion in the posterior root-zone abolishes the knee-phenomenon by its enhancing or depressing influence on the tonus of the rectus muscle.

A. WALLER.

Clinical Cases.

CASES OF ATHETOSIS.

BY FRANCIS WARNER, M.D. LOND., M.R.C.P.

Assistant Physician to the London Hospital, and to the East London Hospital for Children.

THESE cases came under notice at the East London Hospital for children.

Athetosis with Epilepsy.

CASE I.—Alice T., aged 13 years, was brought under my observation in January, 1880, complaining of a swelling in the axilla. This proved to be glandular, and soon subsided. The mother also complained that the left arm had been useless since she was six months old, that she was liable to fits, and rather stupid.

The girl was of fair stature, rather pale and thin, and of weak intellectual power. She could walk, assist her mother in the house, and answer questions, but was very dull and apathetic.

When seen, the following description was taken: *The left upper extremity* is almost useless for most voluntary purposes on account of the condition of the hand. If told to put her hand to her mouth, or to the back of her head, the hand gets there, being moved slowly and awkwardly by the action of the muscles moving the shoulder and elbow, but though flexion and extension of the elbow be sufficiently voluntary to perform such acts, involuntary movements of pronation occur, while the hand is being moved up to the mouth. All through the time of the general movement of this limb, it is obvious that there is much perfectly involuntary movement of the wrist, often causing marked flexion, or affecting independent digits.

As to the condition of the hand, the description was taken while the forearm was gently supported by the mother, the wrist being left perfectly free. It was then noted: The wrist droops, the metacarpus hanging straight down, but frequently supinated rather quickly, but not in a jerky manner, the hand then passes slowly back to its former position, the

series of movements being repeated. Flexion and extension of the wrist through a few degrees are nearly constant movements.

The movements of the fingers are almost incessant, the primary and quickest movements being flexor, followed by slower extension. The movements are slower than the jerks of chorea, and more deliberate, and looking like gesticulations; they are purposeless and gliding in kind.

The fingers are constantly going through grotesque movements, sometimes crossing one another, sometimes one is extended, while the others are flexed; there are no adductor and abductor twitches, such as are usually seen in chorea; the thumb is mostly turned in on the palm, but is often also extended. These finger movements appear utterly purposeless, looking as if the tendons were merely pulling upon dead fingers, as an automaton pulled by strings, or like the movements of a boneless limb, such as an elephant's trunk or an anemone's tentacle. The metacarpal bones are rather contracted together by the arching of the palm of the hand, giving it a narrow appearance.

The muscles of the forearm are neither hypertrophied nor atrophied. The heart and lungs were normal.

The following was the history of the patient.

She appeared healthy at birth: was suckled thirteen months. When six months old, and while in apparently good health, she was taken suddenly in a fit, "as if choking, and wanting to be sick," there was some struggling, and the head was hot; the mother says that only the left arm and leg worked, and that the mouth was drawn to the left. After this fit she lay for three or four days quite helpless. There was no recurrence of fits for some months, then a second similar attack occurred, which was followed, after an interval, by a third paroxysm, each lasting about eight hours. There were in all three attacks during the first two years. There have been no fits from infancy till lately. The left arm has never been healthy since the first fit. The mother cannot say when the movements of the arm were first noticed, but they have certainly lasted many years, probably from infancy. It seems that she had some little use of the hand until the occurrence of the recent slight fits.

During the previous six or eight months epilepsy had returned, two or three slight fits having occurred each month, they set in suddenly, with loss of consciousness.

In the attack she would bite, look strange, and be powerless and stupid after them. Since the onset of the fits she has had less muscular power and more involuntary movements of the left hand as described.

The *patient* is the eldest of a family of six children, four of

whom are living, delicate children, but with no nervous symptoms. One child died in infancy of bronchitis.

The mother has had one miscarriage.

The *father*, aged 40, never had fits. He is now a teetotaller, because he finds that if he takes liquor it makes his head bad. Before the birth of this child he used to drink a good deal, and suffered from it. His mother was liable to fits for many years, falling, struggling, and biting her tongue. Her sister is insane.

The *mother*, aged 34, is liable to hysterical fits, not biting her tongue; she has two sisters, one aged 30, liable to epileptic fits, biting her tongue; the other 22, strong and well.

The patient was under treatment for one month. She took bromide of potassium, and the recurrent fits to a great extent subsided; the girl looked brighter, and the athetosis movements became lessened in degree.

As to causation, the early occurrence of fits in the child, and the inheritance of a tendency to brain disease on both sides of the family, point to heredity as the principal factor in producing disease. The purposeless, involuntary, and gliding character of the movements of the upper extremity were characteristic of athetosis.

Athetosis associated with Chronic Hydrocephalus.

Case II.—Thomas P., first seen, May 7, 1879. The mother complained of the large size of his head, that he could not stand, or sit up, or hold up his head; that he rolled his eyes about very much, and could not use his right hand.

He was then 1 year and 10 months old, obviously hydrocephalic, with a head measuring in circumference 23 inches, and from ear to ear over vertex 12½ inches; eyes much depressed, so that usually a portion of the pupil was hidden. The bones of the calvarium were thin and spread out or expanded, the anterior fontanelle was nearly closed. There were no signs of rickets; he had sixteen teeth. There was no otorrhœa. His general health was good, and no disease was detected in any organ but the brain. The condition of the right upper extremity especially attracted attention; the following description is compiled from notes taken on various occasions.

The limb was useless for any purposive movements; there was no voluntary power over the hand, and but little over the shoulder and elbow. When a flower was held up to the child, he looked at it and made a noise, evidently indicating his pleasure, then moved his left hand to the flower, took hold of it and tried to put it to his mouth. He could not take the flower with his right hand.

The wrist was frequently kept half flexed, while the fingers

were extended and flexed generally all together, in a slow and rhythmical manner. Pronation and supination were not common; the movements of the wrists were usually confined to flexion and extension of the fingers as described.

The hand was moved when pricked. There was no difference between the similar measurements in the two arms, but there was some rigidity of the right arm. The right leg was weak, but presented no athetosis. The child's birth was effected by instruments, and then it was noticed that the head was long and soft at the back. Convulsions occurred the day after birth, then no more till he was four months old, when he was strongly convulsed for four hours and greatly exhausted, but after a day or two he seemed as well as before. At eight or nine months old "he began to feel his feet," then his head was noted as enlarging, and he was frequently slightly convulsed. The mother is sure that before nine months old he could use his right hand, holding a crust in it, and that this power was gradually lost.

The child is still under observation (July 1880); no marked change has occurred except a few fits in June.

Here the condition of the limb was associated with brain disease and convulsions. The movements occurred in a paralysed arm, and were more limited in extent than in Case I.

Double Athetosis not associated with Epilepsy.

Case III.—Eliza R., aged 4½ years, came under observation May 13, 1880. She was a playful, intelligent, pleasing little girl, good and well-behaved, and soon became a general favourite. Her general health was good, and development appeared good in all particulars, but for the athetosis she was well nourished. Her habits were clean, there was no slobbering, and no paralysis, and she never had fits.

The child was unable to stand or do anything for herself on account of the movements of the hands and feet, associated with which there was no doubt some want of muscular power. The muscular condition of the head and face appeared natural. There appeared to be a fair amount of voluntary power over the shoulders and elbows, so that she could hold a doll to her. When told to take hold of a toy she brings her hand to it, but is unable to open the fingers so as to clasp it; all through the time of this effort the fingers are in constant movement. When the object is placed in her hand, she clutches it and is unable to drop it.

Supporting the left upper extremity free from the trunk by holding the humerus, it was possible to observe the following movements succeeding in a rhythmical manner.

The wrist was frequently bent backwards, and sometimes abducted. The thumb was mostly bent in on the palm, turned in under the index finger. The fingers were frequently extended at the metacarpo-phalangeal joints, remaining flexed at the inter-phalangeal joints. The greatest power of extension seemed to be in the index finger. In getting the hand near an object it moved about much before it came in contact with the object, and then could not grasp it on account of the condition of the fingers. The fingers were usually kept flexed; she could not spread them voluntarily. When the child is played with, her fingers spread open spontaneously, as also when attention is specially drawn to the other hand. When one's finger is slipped into her hand, her fingers grasp it, and cannot let it go unless the fingers happen to open of themselves. When she makes an effort and puts her legs out of bed to show her feet, the involuntary movements of the hands and fingers are increased, and movements of pronation and supination are noticeable.

The toes of both feet are continually being flexed and extended, but these movements are less in degree, and less characteristic in kind than those of the hands. She can kick her legs about in bed, but cannot walk.

The eyes and special senses are normal, and general sensation is good. Heart and lungs healthy.

The movements are very strange and purposeless, in kind they have more resemblance to voluntary movements than is usual in athetosis, and less of the gliding, successive-rhythmical character.

During sleep the hands are usually quiet.

This condition of the child appeared to have existed from birth. There were no signs of progressive disease.

The family were quite healthy.

Case IV.—The following case should probably be classed as one of Athetosis of the face associated with *petit mal*.

John Clark came under my observation, May 1878; he was then 12 years of age. The complaint made was that his hands twitched, his face worked much, and that at times he was quite silly.

He was a well-grown lad, of good complexion; his features presented a constrained appearance, and there was much movement of the face. The right hand twitched, but not violently. As he stood up there was some irregular muscular twitching all over the body. He spoke well, and was not himself conscious of the twitching of the face.

The movements of the face were very striking, and were carefully observed. They were principally confined to the parts about the eyes, nostrils and mouth, i.e. not affecting the forehead.

In July, when he was an in-patient at the East London Children's Hospital, I took the following description: Face: complexion somewhat anæmic, mucous membranes a little pale, not markedly so. There is considerable movement of the muscles of the face; he frequently closes his eye, next draws up the angle of the mouth on the same side, then contracts his eyebrows (corrugators), next he elevates them. He appears quite unaware that he is making grimaces. The inner portion of the occipito-fontales (grief muscles) frequently contracts in excess of other portions of these muscles; no movements of the hair or scalp seen.

The orbicularis oculi is frequently contracted, as indicated by the furrows around the eye, and the movements of the lower lid, while at the same time the eye is sufficiently open to allow of the pupil being distinctly seen (coincident of the orbicularis and levator palpebræ ?)

The eyes are much moved in a restless manner, and are frequently turned upwards when the eyelids are being closed. There is never any strabismus. Fundus oculi normal. The tongue is protruded at request, and kept out, and there is much irregular movement of its muscular structure.

As the patient stands upright with his heels together, there is scarcely any movement of the body. When he holds out his hands, and spreads his fingers, they are kept steady, but very slight adductor-abductor movements and almost inappreciable flexor-extensor movements of the fingers are seen; there are also slight movements of the toes. He walks well, with a steady gait, but slight irregular swaying movement of the head.

His heart and lungs appeared normal.

The following was the history of the case. He had always been strong and well till a year and a half or two years previous to my seeing him. He never had convulsions; he went to school when five years old, was bright and intelligent, and did as well as other boys till the autumn of 1876; never any complaint of pain till this time. About that date it was noticed that he made contortions of his face, and frequently nodded his head; then later on the fingers of the right hand began to work. After some months it was noticed that his manner was changed, he became forgetful and vacant—at times as if silly.

When sent on errands he went to wrong shops. He appeared at times to lose himself. One day he was found in the middle of the road at a distance from home, and could not say how he came there or where he lived. He became a tiresome boy, and at times passionate. If beaten he became worse, as if he had lost his reason entirely. In hospital he conversed well, and no mental failure was noticed.

Sleep was sound and refreshing.

As to the inheritance, the father and mother, and the families of which they were members, appeared healthy. The following is the account of the collateral members:—

(1) Male 25 years, healthy.
(2) Male 22 „ „
(3) 5 children died in infancy.
(4) Patient, age 12 years.
(5) Male 9, very healthy.
(6) Female 7, not strong.

The possibility of some defective inheritance is the only probable cause of the boy's illness.

When seen January, 1879, he went to school, was much less absent and forgetful; movements of face were less, principally consisting in contractions of both occipito-frontales muscles. No fits or attacks.

Now (August, 1880) he goes to school; seldom or never falls into "the lost condition." His face is peculiar, and somewhat impassionless, but without pathological movement except in the forehead. As he talks the skin of the forehead is frequently thrown into fine long transverse wrinkles by a slow movement.

Remarks.—These cases appeared to be worth putting forward for the opportunity of contrasting them.

Case I. presented athetosis associated with epilepsy, as is commonly the case. It increased and subsided in severity concurrently with the fits, and there was some mental impairment.

Case II. shewed hydrocephalus and hemiplegia, associated with athetosis and some rigidity. The movements were less in degree than in Cases I. and III.

Case III. presented double athetosis, or double hemikinesis. The cause existed from birth, and the child was free from epilepsy.

Case IV. is classified as athetosis with some reserve; but the persistent rhythmic contractions of the facial muscles, the long continuance of the movements, and general steadiness of gait, suggest a greater alliance to this condition than to chorea. The cases agree in the purposeless, gliding character of the movements which affected principally the upper extremity, and were most marked in the digits, and in the flexor-extensor movements of the wrist, less marked in pronation-supination, and least in the elbow and shoulder.

CASE OF PARETIC DEMENTIA: INTERCURRENT ATTACK OF LEFT-SIDED CONVULSIONS, BEGINNING IN, AND CHIEFLY CONFINED TO, ARM AND FACE; LESION OF POSTERIOR EXTREMITY OF RIGHT SUPERIOR FRONTAL CONVOLUTION.

BY RINGROSE ATKINS, M.A., M.D.

Medical Superintendent, District Lunatic Asylum, Waterford.

THE influence of localised destructive lesions of the motor area of the cortex, and of the subjacent medullary fasciculi, in the production of limited paralyses (crural, brachial, facial, and their associations), is now fairly established by the results of combined clinical and pathological observation; the corresponding effects of irritative lesions of the same area, though recognised and placed on an almost equally firm basis, by the results of experimental physiology, have not, however, as yet received the same amount of extended and definite support, from the experiments of disease, partly from the spreading character of such lesions generally, and partly from the complex train of symptoms to which they give rise; and therefore the particulars of the following case, though not as satisfactory as regards the absolute limitation of the convulsive movements as could be desired, or as is necessary for the purposes of such an inquiry, may yet be of sufficient interest and importance to warrant my recording it, pointing as it does to the localisation of the gross movements of the arm in that region in which experimental investigation has indicated them to be.

John Reilly, formerly a member of the Royal Irish Constabulary, aged 44 years, admitted into the Waterford Asylum May 25th, 1878. The patient, who was unmarried, was accompanied to the Asylum by his brother, from whom it was ascertained that there was no hereditary history of mental or nervous disease, so far as he knew; there was no personal history of syphilis, sexual excesses, or intemperance, the man having been an efficient member of the Force, until struck down by disease. Eighteen months before admission he had had an attack of right hemiplegia and aphasia, from which he

quite recovered; this was, however, followed by a second similar seizure some time after, and again recovery ensued, the man regaining his full powers of locomotion, and, as is stated, of speech also. Five months previous to admission it was noticed that his memory began to fail and his mind to wander, while his utterance became thick and partially indistinct. Gradually he became irritable and developed delusions of persecution, fancying that those around him had conspired to murder him; as a consequence of these delusions he became at times violently excited and noisy, calling loudly at intervals for the police, and in the end becoming so unmanageable and dangerous to those around him, that he had to be brought to the Asylum. When examined immediately on his admission, he was, *mentally*, perfectly quiet and calm, his condition of mind being characterised by dulness, apathy, and apparent indifference to his position and surroundings: he showed no inclination to enter into conversation, and never spoke unless spoken to, and then invariably replied that he felt quite well, and that there was nothing amiss with him; he did not then give expression to any delusion. His facial expression was weak and languid-looking, and the angles of the mouth were slightly drooped. He stood firm and erect, and there was no *motor* disturbance; he walked without any dragging or incoordination of either limb, could stand well with his eyes shut, and turn round when desired without staggering much. The grasp of the right hand was not as powerful as that of the left, but the difference was not great. When replying to questions, there was a noticeable tremor and jerky action of the lips, and he evidently found difficulty in completing his words, as the last syllables and latter words of a sentence were drawled out and jumbled together: the tongue was affected with tremor and spasmodically protruded and retracted. The pupils were equal and normally motile. There was no apparent defect of vision or hearing. The condition of *tactile* sensibility was not investigated, as the degree of dementia was too deep to allow of reliable results being obtained; he had, however, two sores over the buttocks, which he did not appear to feel, or hardly to be conscious of, so that it is probable that there was analgesia to some extent at any rate. His general muscular development was good. During his residence in the Asylum the mental powers rapidly failed, the loss of memory increasing and the dementia deepening, while the articulatory defect steadily progressed. With this, however, there was no decided loss of motor power in any of the limbs, beyond a general muscular weakness and unsteadiness; and he was in this condition on the 23rd of September, 1878, when, at 11.15 A.M. on that day, he was suddenly seized with unilateral left-sided

convulsions, and became unconscious. The spasms—which were clonic—began in the left arm, as a whole, and in the left side of the face (angle of mouth and forehead), and travelled downwards to the leg, which was much more mildly convulsed. Soon after the spasmodic movements commenced in the same order at the right side, but were neither so powerful nor continuous, soon ceasing there and in the left leg. The left arm and left side of face continued, however, to work at very short intervals, the movements involving the entire arm from the shoulder down, the limb being jerked up and partially thrown across the chest, the angle of the mouth twitching at the same time. At 12 o'clock, the spasmodic movements continuing uninterruptedly, forty grains of bromide of potassium were administered, and at 3 o'clock, but little abatement being noticeable, another dose of forty grains was given (note made at 3.30), and at 4.20 P.M. the fit ceased, and he passed into a heavy sleep, in which he lay at 11.20 P.M. The next day it was with difficulty he could be roused, and for some days after he was drowsy, and the dementia had perceptibly deepened. From this time there ensued a more distinct loss of motor power, the left side being chiefly affected, and especially the left arm; the speech defects also rapidly increased, the lips trembling and jerking to a marked degree whenever he attempted to speak, which he rarely did: he used to keep the jaws tightly clenched, and was almost constantly grinding his teeth. He had no further convulsive attack, but becoming more and more weakened and demented, he sank and died on January 3rd, 1879.

Autopsy forty hours after death. — The body was fairly nourished, the calvarium dense and solid throughout, the diplöe being replaced by compact bony tissue. There were no morbid adhesions between the dura mater and the inner surface of the skull. The membrane was unaffected, its vessels posteriorly were filled to repletion with blood (hypostatic congestion), and it was attached by a few filamentous bands to the pia mater at several spots parallel to the great longitudinal fissure. The pia-arachnoid was somewhat opaque, especially where it bridged the sulci, being here of a milky colour. At a spot corresponding to the posterior extremity of the superior frontal, and the summit of the ascending frontal convolutions of the right hemisphere, the membrane was distended into a bulla the size of a walnut, by a collection of fluid beneath; and a similar bulla, but much smaller, occupied a more anterior position on the left superior frontal convolution. The membrane enclosing the fluid was thin and pellucid, and on being ruptured the contents were found to consist of a clear reddish-brown serum. On carefully removing the pia mater from the

spots thus affected, a deep atrophic depression, large enough to admit the tip of the thumb, was found involving the posterior extremity of the right superior, and portion of the summit of the right ascending frontal convolutions; and a slight depression existed beneath the bulla on the left superior frontal. There was no erosion of the surface, or softening of the substance of the affected convolutions, the cortex being apparently only depressed into a pit-like hollow. The pia mater, though adherent to the cortex in small patches scattered over the fronto-parietal convolutions, was not so at the atrophied spots, the membrane stripping readily. There was no gross lesion in any other part of the cortex, and no morbid conditions discoverable by the naked eye existed in the medullary substance or central ganglia. The vessels at the base were slightly atheromatous. Portions of the atrophied and neighbouring convolutions, together with the pons medullæ and a piece of the cerebellum, were placed aside; and having been hardened in iodised spirit and bichromate of potash, prepared sections of the atrophied gyri showed the following morbid conditions:—The cortex did not appear to the naked eye to be notably thinned. The walls of its *vessels* were greatly thickened by a closely-set nuclear overgrowth, so that their laminæ were contracted often to a very considerable extent. Viewed in transverse section, the fibrous coat of the arterioles, thickened and wavy, remained in some instances otherwise intact, the aggregation of nuclei surrounding it and distending the investing sheath; while in other instances the nuclear growth seemed to have taken possession of the entire thickness of the wall, either obscuring or altogether replacing the normal tissues. Viewed longitudinally, the surface of the arterioles appeared to be covered over closely by the nuclei. The perivascular spaces were comparatively clear and did not contain scattered nuclei, particles of hæmatoidin or granular débris. Sections through the adherent pia mater and cortex showed the membrane to be much thickened, its meshes infiltered with nuclei and the remains of minute blood extravasations, the walls of its vessels coarse and thickened, though not nucleated in the same way as those in the subjacent cortex, and their lamina filled with broken-down blood-corpuscles. Where firmly adherent to the cortex, the latter seemed to be continuous with the membrane by a fine granular material in which the entering vessels were embedded; and where removed purposely, or in the preparation of the sections, the cortical surface was irregular and overlaid by a refractive yellowish material like disintegrated blood. The *nerve-cells* in all the sections examined seemed to be similarly affected, any difference being one of degree; the different cortical

laminæ appeared to be compressed, and the first layer was coarse and molecular-looking, and studded with many free nuclei and ovoid refractive bodies (artificial products?). The larger corpuscles of the third layer were diminished in number in patches, a group appearing, and then a blank occurring, with but one or two; many of those remaining had lost both basal and apical processes or basal only, the angles being rounded and the entire corpuscle surrounded by a clear space. Few were deeply pigmented, or in an absolutely granular condition. The majority of the smaller cells seemed to be unaffected. The *neuroglia* throughout was coarse and molecular, containing lymphoid cells, granular bodies, and refractive spots.

The partially localised convulsions recorded above were clearly intercurrent in the course of a case of general paralytic dementia, of which this was, I believe, an example of that dubious form of the disease met with in this country; and it is a question, of course, how far the cortical lesion can be considered as playing a part in the causation of such seizure. Unilateral or partial convulsions, as is universally known, are of common occurrence in the course of general paralysis, though it was not until a comparatively recent period that their cortical origin began to be recognised and investigated; and even now, while it is conceded by the most recent authorities on the subject that the localised adhesions of the pia mater to the cortex in the motor area are the pathological factors in the production of the limited convulsions and spasms, there are not, I believe, as yet many cases on record where any single lesion of this kind, in any definite motor centre, has been directly associated with any limited convulsive seizure in the relation of cause and effect. In the present instance the most pronounced and definite cortical lesion, though not an adhesion, occupied a position closely corresponding to that which Ferrier has experimentally shown to be concerned in certain movements of the arm, and which is supported by the facts of human pathology. He quotes a case recorded by Lépine, of left hemiplegia followed by convulsions limited chiefly to the left arm, in which a small hæmorrhage was found at the posterior extremity of the first frontal convolution of the right hemisphere; and another, by the same observer, of left hemiplegia, in which convulsions occurred, either limited to the left arm or invariably commencing there; a yellow patch was found at the bases of the first and second frontal convolutions of the right hemisphere. Though not strictly analogous to these cases, either in the sequence of the symptoms or in the nature of the lesions found *post mortem*, the observation which I have recorded possesses, in common with them, the

essential features of limited convulsions and localised cortical disease, and hence it may be regarded as bearing on the question at issue. It may, perhaps, still be asserted by some, that such convulsions coming on in this way cannot always be traced to their origin, that they occur independently of gross or even of discoverable microscopic changes in the brain structures; but though this is doubtless true in certain cases where the seizures are of a more distinctly epileptic type, yet I think that their occurrence once in the course of such a case, the limited character of the convulsions at the beginning and termination of the attack, and the duration of the spasmodic movements, point strongly to their purely organic origin; and though the lesion itself may not, when discovered, have presented the characteristics of an "irritative" one, yet having regard to the time which elapsed since it had been active and the subsequent changes which may have occurred to alter its appearance, the position it occupied should, I think, be considered rather than its exact nature. So impressed was I, indeed, at the time with the character of the convulsive seizure, that I ventured the opinion that it might be possibly due to some lesion in the motor area of the cortex; and any other condition found failing to account for it, the morbid alteration affecting the posterior extremity of the first frontal and the summit of the ascending frontal gyri must, I believe, be credited with a causal influence in the production of the limited convulsions.

A CASE OF INJURY TO THE LEFT FRONTAL LOBE OF BRAIN, FOLLOWED BY AN ABSCESS.

BY HENRY ASHBY, M.D. LOND.

Physician to the General Hospital for Sick Children, Manchester.

J. H. W., aged 6 years, was admitted to the hospital Jan. 4, 1881, with the following history. He is the youngest of nine healthy children, and was well till July 26, 1880. On that date, while he was playing with his sister, he fell forward upon a sharp pitchfork she held in her hand, the prong striking him above the left eye. No external wound resulted, except an abrasion of the skin, the upper eyelid became much swollen, and he suffered much pain in the forehead and ears. During the next few weeks he was irritable, thirsty, feverish at night, and frequently vomited. Just before Christmas his sight failed him, in consequence of which he was brought from Huddersfield to the Manchester Eye Hospital for advice, and was transferred to the children's hospital.

On admission.—He is an irritable child, fairly well nourished, talks with a nasal twang, and is apparently somewhat deaf, or dull of comprehension. There is no mark of any wound about the left eye, but he screws it up. He is quite blind; the pupils are dilated, but act sluggishly. There is slight but well-marked right facial paralysis.

He lies in bed with knees drawn up: he can use both hands, but the right is the weakest; on being told to squeeze one's hand, he exerts some power, then says "I can't," and cries. He can walk fairly well, but is weak on his legs. All the superficial reflexes well-marked. Knee reflex, present not exaggerated; no rigidity of the limbs. Both optic discs swollen; edges indistinct; veins tortuous and full; arteries only seen on discs here and there as fine threads; several yellow spots on discs obscuring vessels, and others glistening in retina, near disc (hæmorrhages).

He remained much in the same condition, being irritable, fretful when touched, and sometimes crying out at night, and passing his urine and fæces into the bed.

January 28th.—Had a fit this morning, lasting twenty minutes, clenching his hands, and flexing limbs; he became comatose, and died in the evening.

Autopsy.—Forty-eight hours after death. Head only examined. On removing skull and dura mater, it is apparent that the convolutions of the left frontal lobe are dry on the surface and much flattened out, the whole width of the lobe being increased. The ascending frontal, superior, middle, and inferior frontal convolutions are involved in the flattening; the ascending parietal in lesser degree.

On gently raising the frontal lobe from its bed, it is evident from its elastic feel that it contains fluid, and from the yellow tinge of the inferior surface that this fluid is pus. It is adherent to the centre of the internal surface of the roof of the orbit, at a spot the size of a threepenny piece. On removing the brain and examining the bone, there is a hole through the roof of the orbit, large enough to admit the tip of the little finger, directed backwards and upwards. On section of the frontal lobe, it is found to contain four to five ounces of thick greenish pus; the inferior wall of the abscess is not much thicker than cardboard; the pus has evidently exerted considerable pressure on the inferior, middle, superior, and ascending frontal convolutions, flattening them against the roof of the skull, and involving the white substance immediately beneath them, but leaving the whole thickness of their grey matter. Examination of the optic discs shows that they are prominent, and the veins are full and tortuous. The optic sheaths behind the eyeballs are in a semi-distended state. A naked eye examination of the nerves, between the fundus and chiasma, after hardening in ammonia bichrom. and spirit, shows them to be swollen (in comparison with a healthy nerve). Longitudinal sections of the fundus, examined microscopically, show the disc much swollen, increase of nuclei, small collections of nuclei around arteries, and the lymph channels between the strands of the nerve, much distended. Transverse sections made at intervals to chiasma show these enlarged lymph-spaces in the nerve for $\frac{1}{3}$ to $\frac{1}{2}$ in. behind fundus; nearer the chiasma the nerve was apparently normal. Both nerves exhibit these changes.

Remarks.—In this case it appears that the sharp, narrow prong of the pitchfork entered the orbit, between the upper eyelid and eyeball, pierced the roof of the orbit, and entered the frontal lobe for a distance apparently of at least an inch, judging from the size of the opening through the orbital plate. This injury was not immediately fatal, but was followed by a local inflammation, which ended in an abscess that

proved fatal in six months. The abscess totally destroyed the convolutions on the under surface of the frontal lobe, pushing inwards and undermining those on the inner side, thrusting upwards and flattening out the ascending, superior, middle, and inferior transverse, against the roof of the skull, completely destroying the white matter underlying them, and leaving the whole thickness of their grey substance, but in a state of compression.

Notwithstanding this state of things, the boy could walk to hospital, his memory and general intelligence was good, and there was no loss of speech. But he was somewhat deaf; had slight facial paralysis on the opposite side, and some weakness of the right arm; troublesome pains along the facial divisions of the fifth, and loss of control over his sphincters.

PAIN IN THE OCCIPUT AND BACK OF NECK. STRONG RETRACTION OF THE HEAD. GLIOMA OF THE RIGHT TEMPORO-SPHENOIDAL LOBE. NO DISEASE OF CEREBELLUM.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

THE patient was a gentleman aged twenty-seven. In 1873, as I am informed by his medical attendant, he complained of what he called "giddiness" or "nervous attacks." He subsequently spent several months in North America, engaged in sporting. In 1877 his symptoms were nearly the same as in 1873, and a physician who saw him regarded these and the former attacks as those of *petit mal*. He never lost consciousness. During last autumn he spent the season in successful grouse shooting. A week before his death he hunted on one day from ten to six o'clock. During the last two months of his life he complained of severe pain over the occiput and back of the neck, extending upwards on the right side of the head to the forehead and sometimes down the neck. The pain generally came on during the night, and disappeared after breakfast. He was sometimes free from pain for several days together. There was occasionally a little sickness. In the paroxysms of pain the head was drawn back during the last few weeks of his life, and he complained of hemiopia. There was very slight facial paralysis of the left side.

Autopsy.—There was nothing abnormal in the appearance of the cerebral surface on opening the skull. When the brain was removed and turned upside down, it was noted that the sulci of the inferior surface of the right temporo-sphenoidal lobe were effaced. Palpation of this lobe was accompanied by a sense of fluctuation, in striking contrast with the corresponding lobe of the left side, which presented the ordinary resistance of healthy brain. A vertical section through the right temporo-sphenoidal lobe showed that the grey matter of the cortex had disappeared, the exposed walls of the section presenting a flesh colour. The structure was, in parts, of a consis-

tence somewhat more dense than that of healthy medullary matter which, however, it a good deal resembled in appearance. At the depth of rather less than an inch the knife passed into a cyst-like cavity containing about two drachms of straw-coloured fluid, and large enough to hold two or three walnuts. The walls of the cavity were very smooth, and consisted of the same flesh-coloured substance as had been previously cut through. Several small hæmorrhages were observed in the walls of the section. The abnormal structure or tumour merged quite insensibly into the surrounding brain substance, which was more or less softened throughout the whole of the right temporo-sphenoidal lobe, and some distance into the occipital lobe, as well as, though to a less extent, into the lower part of the convolutions bordering the fissure of Rolando.

The cerebellum presented no signs of disease in any portion of its structure, nor was there any appearance of flattening of its upper surface. Under the microscope the tumour presented the characters of soft glioma tending to myxoma, numerous protoplasmic cells, with a few much larger ones, containing many nuclei, and bearing a general resemblance to mucus globules.

The patient was only seen by me shortly before his death, when he was in a state of coma, which was sometimes profound, whilst at other times he could stand, talk, and swallow liquids, although with great difficulty. The coma was partly due to a dose of morphia, which had been given for the relief of severe pain. The head was retracted, so that the occiput was nearly touching the cervical portion of the spinal column, the eyes were upturned and divergent, there was slight facial paralysis of the left side. He would keep his right hand to his forehead, and when asked if he was suffering pain in the head, would signify that this was so.

I learned that for upwards of two months past he had been subject to daily attacks, lasting many hours, of severe pain about the back of the head and neck, accompanied occasionally, but not often, by vomiting. In these attacks the head was drawn back, I was informed, in the way which I have described. Impairment of sight had been complained of, and there had apparently been hemiopia. An ophthalmoscopic examination had been proposed, but not made.

Incomplete as is the case, it seems an important one to place on record, because of the very striking symptom, retraction of the head.

Dr. Hughlings-Jackson has recorded examples of what he calls "cerebellar rigidity (part of which is retraction of the head), in cases of cerebellar tumour. In some of these there were,

besides, paroxysms like those of surgical tetanus. Similar observations have been made by Dr. Stephen Mackenzie. In the 'Lancet,' January 24, 1880, Dr. Hughlings-Jackson writes: "I have only once seen seizures like tetanus in a case of local cerebral disease, and in that case they occurred during the last hours of life—during dying." In the paper referred to he records a case of retraction of the head with tumour of the cerebellum. Of course retraction of the head is known to occur from other pathological changes, as cerebro-spinal meningitis for example, but for purposes of localisation cases of tumour supply the best evidence, though, unfortunately, from their size this evidence is not often very definite.

It is remarkable that a person suffering from so extensive and grave an intra-cranial lesion should have been able to enjoy a long day's hunting within a week of his death.

Abstracts of British and Foreign Journals.

On Hydrops Articulorum intermittens.—Under the above heading Dr. Seeligmüller has published in the *Deutsche Medicinische Wochenschrift* (1880, Nos. 5 and 6), a description of a case of this obscure and interesting affection, followed by an analysis of 12 observations of the disease made by other authors, this being the complete literature of intermittent swellings of joints. Seeligmüller gives the following definition of the disease: "An otherwise healthy adult is affected in regular intervals, generally without any prodom and without discernible cause, by *an intense swelling of one or both knee-joints, there being no signs of inflammation or fever.* The swelling reaches, after a certain time, its maximum (stadium incrementi), remains there for a while on the climax (stadium acmes), and disappears likewise after some time, without leaving the slightest alteration in the affected joint. (In Seeligmüller's own observation, however, this was not the case; the left knee was always a little swelled and painful to the touch.) *In most cases the swelling returns with mathematical precision on a certain day,* sometimes even at a fixed hour, so that the patients can foretell the outset of the affection."

Since the publication of Seeligmüller's article, three other cases have been reported in the same journal by Pletzer (No. 3 f. 1880), Fiedler (No. 3, 1881), and Kolbe (No. 4, 1881).

Concerning the *etiology* of the affection, out of 13 cases collected by Seeligmüller (including his own observation) there were 7 females and 6 males. The three cases published since were all females, my own patient (of which I shall speak further on) also female. This gives a total of 11 females to 6 males.

The *age* of the patients was between 12 and 54 years; my patient was 9 years at the time when the disease was first noticed.

As to the *cause* of the affection, in two cases there is a history of intermittent fever. Kolbe's patient is evidently hysterical. In his, as in two other cases, an injury of the knee must be considered as the immediate cause of the disease.

The intervals between the fits are very variable; 8 days, 11 days, 13 days, 4 weeks. In my case they are absolutely irregular. The case reported by P. Bruns is most remarkable in this respect, as the patient had the swelling at regular intervals of 12 days during 8 years. In many cases there was an absolute intermission of months, and even years, after which the affection began again, sometimes adopting a new type. The duration of a fit is about from 3 to 8 days; in my case sometimes 2 to 3 weeks.

In 9 out of Seeligmüller's 13 observations, the knee-joint was the only articulation affected by the disease; for this reason it was always described as hydrops genu intermittens. Twice there is no particular mention as to the seat of the swelling. One patient (Löwenthal's case) began with swelling of the right elbow. In Seeligmüller's case, the left knee and one of the hip-joints showed the swelling. In Fiedler's case, as well as in mine, nearly every articulation of the body is occasionally the seat of the disease, but there is usually only a single joint affected in each attack, sometimes two at a time.

There is no notice of any *secondary alteration* of the joints, not even in Seeligmüller's case of 30 years' standing.

The history of my case of Hydrops articulorum intermittens is this:—

Marie M., æt. 11, has been suffering for the last two years from swelling of one or another joint, combined with extreme pain, hyperæsthesia of the skin in the neighbourhood of the swelled joint, consequently entire loss of function during the attack. The onset of the swelling is as sudden as a stroke of lightning; no symptom of any kind precedes the attack. For instance, the child is writing; suddenly she feels a violent pain in her wrist, and is unable to finish the word that she was just going to write down. The swelling increases more or less during the next 2 or 3 days; it remains in the stadium acmes for a variable time, and finally disappears gradually.

At first the attacks occurred with intervals of several weeks; but, by degrees, they came more frequently and lasted longer. Since February 1880 there were only intervals of a few hours, so that the little patient could hardly ever leave her room. The pain was so great that even when only a joint of an upper extremity was affected, the girl scarcely ventured to walk about in the room, because the commotion caused thereby increased her suffering. The disease has some seats of predilection, viz. the wrist, knees, and the ankle-joint; but all articulations of the extre-

mities, and sometimes the cervical vertebræ, suffer occasionally. Fever or any other disturbances of general health were never observed.

The *treatment* consisted at first in anti-rheumatic remedies: salicylic acid, quinine, iodide of potassium, colchicum; hot steam-baths were followed by an evident increase of the disease, while other remedies had no effect of any kind. Several other drugs were tried with the same negative result. The first thing which seemed to be of some use was arsenic. While this remedy was given, the child had an interval of 6 days; but it must be mentioned that she was then staying in the country for a few weeks and could be in the open air nearly all day, so that this may partly account for the improvement.

I saw the patient for the first time on the 18th of August, 1880, and began at once with applications of the galvanic current. I omitted to say that faradic electricity had been used in the beginning of the illness by the father of the child, who is a physician in Dresden. At first it seemed to alleviate the pain a little, but after awhile it failed to do so, whereas the application of an ice-bag had a better, though but a temporary effect. In examining the child I stated that the local use of a faradic current *increased* the pain, without in the least relieving the swelling. This is a remarkable fact with regard to the distinction of this disease from rheumatic inflammation of the joints. In these cases, as has been shown by Drosdoff, Abramowosky, and frequently confirmed by myself, there is a considerable diminution of the faradic sensibility in the skin of the affected parts, so that the strongest currents may be applied without pain; besides faradisation is always followed by more or less alleviation of the pain and swelling.

My treatment consisted in the application of a moderately strong galvanic current to the cervical region of the spine during five minutes daily. I intended to act on the vasomotor centres situated in those parts. The effect of this proceeding was decidedly good. Within a few weeks the free intervals became longer; the swelling, when it occurred, was considerably less intense, the pain tolerable. The child began to go to school, which she had not been able to do for several months. In January there was an interval of 22 days, since then till now (March 5th) the patient has had two very slight attacks, and lasting a few days. I must not forget to add that since the end of November the child recommenced to take arsenic, but in very small doses (3 drops of Fowler's solution in a day). I think it may be hoped that a per-

severing use of the galvanic current, occasionally combined with arsenic, will cure the little patient of her obstinate and troublesome complaint, and if a conclusion ex juvantibus be permitted, the undeniable action of the electric current cannot fail to support the idea expressed by Seeligmüller and other authors, that Hydrops articulorum intermittens must be considered as a chronic affection of the vaso-motor nerves.

R. H. Pierson, M.D.

Löwenfeld on Electro-Therapy of the Brain.—Löwenfeld (*Centralbl. f. d. med. Wissenschf.* No. 8, 1881), gives a preliminary account of the results of experiments he has made on rabbits and other animals as to the effect of passing electrical currents through the head longitudinally or transversely; and also of placing one pole on the exposed brain and the other on the neck, according to the method of Legros and Onimus. He finds—

1. A descending current (+ on forehead, − on neck) causes contraction of the vessels of the pia mater.

2. An ascending current (+ on neck, − on forehead) causes dilatation of the vessels.

3. In transversely directed currents, there is dilatation of the vessels in the side of the anode, and contraction at the cathode.

4. Induced currents, longitudinally or transversely directed, cause hyperæmia of the brain. This point requires further investigation, but it seems as if the induced current is not limited to action on the vessels.

Gombault on the Segmentary Periaxillary Neuritis.—In the *Archives de Neurologie*, No. 2, 1880, Gombault finishes his paper on segmentary neuritis, the first part of which was noticed in 'Brain,' Part XI. p. 423. Gombault is of opinion that the periaxillary neuritis may result in Wallerian degeneration when the inflammatory process is sufficiently intense to cause rupture of the axis cylinder. This occurs from a participation of the axis cylinder itself on the inflammatory action, not by mere pressure of inflammatory products. He draws an analogy between the periaxillary neuritis proper, and the Wallerian degeneration which may accompany it, and the changes which Ranvier has shown to occur in the proximal and distal ends of a divided nerve respectively. In the former there is a parenchymatous neuritis, in the latter there is degeneration in consequence of the separation of the nerve from its trophic centre. In traumatic neuritis in man the changes are such

as are seen in periaxillary neuritis. In chronic anterior polio-myelitis the question is whether the changes which occur in the nerves are merely passive, and merely the result of the disappearance of the multipolar cells, or whether there is active irritation or inflammatory action. The latter is the view advocated by Charcot, and Gombault is of opinion that the facts are in favour of it. Periaxillary neuritis exists, and the irregularity of the distribution of this process sufficiently accounts for the appearance of degeneration at different levels in different nerves.

As regards the general action of lead on the lower animals it appears to induce all the principal phenomena of anterior poliomyelitis. The sensory nerves do not seem to be affected. In the spinal cord the anterior horns alone are affected. The multipolar cells are in large measure vacuolated; the neuroglia and walls of the vessels indicate products of inflammatory irritation. In the muscles also of guinea-pigs poisoned with lead, very delicate fibres are visible, not met with in normal guinea-pigs.

Debove and Boudet on the Pathogeny of Tremors.—Debove and Boudet de Paris (*Archives de Neurologie*, October 1880) apply the results of an investigation of the characters of the muscle curves in healthy individuals under varying conditions to the explanation of tremors.

The sudden elongation of a muscle excites it to contraction (the tendon phenomenon), and this may be brought about by striking its tendon, mechanical traction, or by the contraction of its antagonist. Thus the curve of the action of the biceps by a single stimulus is not a single curve, but a double one, the second elevation being caused by the reflex contraction due to the action of the antagonist triceps. If the traction applied to a muscle is persistent, with variations in its intensity, such as may be induced by traction on a band of caoutchouc, a series of reflex contractions are induced in the muscle, rhythmical in character and almost constant for each muscular group (10 to 12 per second). Fatigue does not alter the number, but affects the amplitude of the oscillations.

A muscle in a state of contracture acts like the elastic band of varying tension, in relation to the antagonist muscle. Set in action by volitional impulse, by electric stimulation, or by the weight of the limb, it excites rhythmical oscillations in its antagonist which constitute tremor.

Senile tremor and paralysis agitans are explained by the presence of muscles in a state of contracture. The weight of the limb itself

may keep up constant trembling, and the authors have found that spontaneous tremors can be checked, or greatly diminished, by immersion of the patient in a bath, which has the effect of removing the weight of the limbs.

Bourneville and d'Olier on the Pathology of Epileptic Dementia.—Bourneville and d'Olier, assisted in their microscopical researches by Brissaud, give the clinical history and post-mortem appearances in a number of cases of epileptic dementia observed at the Bicêtre. (*Archives de Neurologie*, October 1880.)

In a large number of cases there are no special morbid appearances discoverable post-mortem.

In others again there are well-marked lesions, of the general character of diffuse encephalitis. The seat of these lesions is almost exclusively in the frontal lobes. Their situation here accounts for the absence of paralysis, and the dementia observable.

The lesions are somewhat similar to those found in general paralysis, but they differ in certain points. No miliary aneurisms are discoverable, and, while in general paralysis of the classic type the lesions affect the superficial layers of the cortex, in epileptic dementia, the lesions affect more particularly the deeper layers and adjacent medullary fibres, which appear yellowish to the naked eye. The symptoms which characterise this epileptic dementia differ also considerably from those of general paralysis. It is only in the later stages that the two resemble each other.

There are no extravagant notions, or changes of character, &c., nor inequality of the pupils such as are seen in the early stages of general paralysis. It is a form of dementia intimately connected with epilepsy. The patients differ completely from those, a few in number, who in addition to epilepsy become affected with general paralysis. In such cases the general paralysis exhibits its usual characters. Two cases of this kind under observation establish the fact beyond all doubt.

D. Ferrier.

Rosenbach on Reflexes in Sleep. By Dr. Ottomar Rosenbach. *Zeitschrift f. Klinische Medicin*, 1881. Bd. I. Hft. 2. —Dr. Rosenbach remarks that the various reflexes have not been hitherto systematically tested during sleep. We admit in a general way that the sleeping organism is, as regards its reflex activities, in the condition of a decapitated animal, but, says he, the condition is not quite so simple, and varies greatly

with the depth of sleep, of which we have no exact standard. It is in fact by the reflexes themselves that we are to estimate the depth of sleep, as that of coma. On children, as being the most suitable subjects, he systematically observed the reflexes excited by tickling the palm of the hand, the sole of the foot, and occasionally other parts of the body, also the reaction of the pupil, the cremasteric reflex, the abdominal reflex, and the tendon reflex. He states that at bedtime, or when children are sleepy, the reflex excitability is increased, until they suddenly "set" in the position they happen to be in—a stage of restlessness with which he considers the convulsive stage of chloroform narcosis to be comparable. He says that the first period of sleep, which is light, is characterised by weakening of the reflexes and commencing contraction of the pupil; that in the second or deeper period, the abdominal and cremasteric reflexes and the tendon reflex are abolished; that in the third period or deepest sleep, tickling of the sole of the foot, of the nose and of the ear still liberates reflexes. To the question whether the activity of the sphincters in sleep and in coma is effected by a direct cerebral condition of excitation, or whether it is reflex, he opines that probably there is an augmented cerebral activity protective of the outlets of the sleeping organism. He argues that if sleep were but a cerebral paresis, we ought to get enhancement of spinal reflexes in sleeping, as in decapitated animals; whereas we see that the reflexes, especially the abdominal reflex, weaken as sleep deepens, and that later, various irritations are capable of eliciting precise reflexes. Hence the supposition that not only is there a temporary emancipation of the cord, but at the same time some kind of active restraint of the reflex activities. The author remarks that we may suppose this influence, acting presumably along motor paths, to be strongly exercised in disease, in proportion to its suddenness; thus it should be strongest in traumatic and in experimental paraplegiæ, while slowly acting lesions should bring out the opposite or reflex enhancing influence.

A. Waller.

A Definition of Insanity. Dr. G. M. Beard.—Dr. Beard, in a paper read before the New York Medico-Legal Society, gives the following definition of insanity:—

Insanity is a disease of the brain, in which mental co-ordination is seriously impaired. He claims for this definition that it is a

short description, including all real cases of insanity, and excluding all those that are not insane. Dr. Hammond objected to the statement that insanity is a disease of the brain. To this Dr. Beard replies that by the expression he means that insanity is one of the results or manifestations of disease of the brain. By the use of the word "seriously" he excludes the lighter forms of trance, the temporary and slighter influence of alcohol or opium, &c. If these be prolonged and severe (e.g., the delirium of fever), he would regard the person so affected as insane. Dr. Beard looks to the future to decide the question of the legal responsibility of cases of alcoholic trance.

With regard to the meaning of mental "co-ordination," he is unable to define the word; but believes that "all persons sufficiently intelligent to discuss the subject would agree in their definition could they frame their ideas in language."

The word "responsibility" stood in the place of "co-ordination" in the first form. Dr. Beard gave his definition, and he would still use the former phrase in a court of law.

To meet the objections that naturally arise in regard to the vague and indefinable terms in which his definition is couched, he replies that insanity is a vague condition, a disease of gradations; that only an approximate statement of its nature is possible; and that this statement will be relative to our knowledge of the subject.[1]

Dr. Beard concludes with the following propositions as the outcome of the Gosling case, in which the patient was declared to be labouring under general paresis by all the specialists on insanity who testified in the case, but was allowed to go free by the Court, two general practitioners having testified to his sanity.

First. It showed the need of a definition of insanity.

Second. It illustrated the difficulty of the diagnosis of insanity, the need of repeated visits, so as to examine his behaviour in what may be a remission of the disease.

Third. The interest attaching to the predisposing and exciting causes of insanity.

[1] The first step in accepting a definition is to form a rational conception of what it means and to translate it into other forms of expression. But Dr. Beard admits the impossibility of doing this, and relies on his hearers interpreting the meaning, though all are unable to express themselves in words. It would be of little use then to define insanity in this manner in a court of law, where an immediate explanation of the scientific term "co-ordination" would be demanded. Is this phrase more descriptive than "mental adjustment" or "personal equation," or indeed, is it equivalent to saying more than "insanity is insanity"?

Fourth. The need of distinguishing between the value of the testimony of experts and non-experts.

Fifth. The diagnosis of insanity should be purely psychological, and not at all physical, by contrasting the mental manifestations of the individual in health with the mental manifestations of the same individual in disease, and also with the mental manifestations of the average man in the same condition of life. If a diagnosis of insanity cannot be made out by the mental manifestations alone, it cannot be made out at all.

Sixth. A clear and scientific confession of ignorance should be made, if necessary.

Seventh. Experts should be called and paid by the Court, and there should be a general reform of lunacy jurisprudence.

[It is evident that the fourth, fifth, sixth, and seventh of the foregoing propositions deal with the legal rather than the scientific aspects of the disease. In another place Dr. Beard says, "We may be justified in condemning a man scientifically, when we have no right to condemn him legally;" and again, "The law makes inquiry only of legal irresponsibility as the result of mental incoordination."]

A. R. URQUHART, M.D.

Tassi on a successful Case of Trephining.—Dr. Eugenio Tassi was more successful with his patient. R. P., a wandering merchant, fell asleep upon a wall and lost his equilibrium, striking a pebble on the ground with the left side of the head. He was found unconscious, and was brought to the hospital, when it was found that he had a contusion of the scalp and a fracture at the posterior part of the left frontal bone, with symptoms of concussion of the brain. Consciousness returned; the wound did well for eight days, and there was no constitutional inflammatory action, when one morning it was found that he was paralysed in the right arm and leg, and in a comatose condition. It was determined to use the trephine, which was done thirty-two days after the injury. It was applied "in correspondence with the coronal point (punto stefanico) between the pteric and the bregmatic points." The pia mater was intact; but several detached fragments of bone were found pressing upon it, which were cautiously removed. While this was being done the arm was noticed to move a little. In a few days the mental powers were completely re-established, speech returned, and the limbs recovered their lost activity. During the treatment

there was a spontaneous luxation of the right thigh, the cause of which could not be ascertained. The patient left the hospital completely cured, though he had to use a crutch, owing to the dislocation.

Seppilli on a Case of Atrophy of the Cerebellum.—In the *Rivista Sperimentale di Freniatria* (Anno V., fascicolo iv., 1880) there is a long article on this rare disease by Dr. Seppilli. The patient, a woman of feeble intelligence, had at the age of twenty-eight been afflicted with typhoid fever. During her convalescence there was observed a general tremor and disorder in all the motions of the body. This was accompanied by distinct disturbance of mental power, incoherence of thought and a proneness to weeping and laughing, with hallucinations of sight and hearing. She thought she saw beasts, which terrified her, and heard voices calling to her. This mental disturbance and disorder of motion increased till she was brought into the asylum of Reggio Emilia.

On entering it was found that all the muscles of the face, eyes, and tongue were disordered with clonic contractions. The muscles of the neck were rigid, the arms bent and contracted, and were extended with difficulty. There were occasionally rapid ataxic movements of the arms with contortions of the fingers. The legs were semiflexed and slightly contracted. The general sensibility seemed increased, and tendon reflex of the patella was easily brought out. These disorderly motions increased with the mental excitement, declined with mental calm, and ceased during sleep. A period of excitement alternated with a period of quiet which passed into stupor. The articulation was much hindered by the motor disorders, which were greater at one time than at another. The patient gradually wasted away, and died nineteen days after admission.

The principal abnormality found was the smallness of the cerebellum, which only weighed 56 grammes, less than half the usual weight in women. It was regular in its shape, but hardened in texture, and the grey matter was much wasted. The pons, crura and medulla oblongata were of the normal size. The convolutions of the cerebellum were small and unequal. In the texture of the organ the cells of Purkinje were much less numerous than usual, and those which were found had only half the usual size, and their processes were small and thin. In the median lobe the cells of Purkinje were more abundant, though not so frequent as in a

normal cerebellum. The grey matter of the nucleus dentatus was much diminished. The connective tissue was in excess, and the walls of the meningeal vessels thickened.

The general result might be said to be atrophy with sclerosis limited in great measure to the lateral lobes.

He notices the observation of Stefani, that in pigeons the cellules of Purkinje are found to be atrophied after lesions of the semicircular canals.

Dr. Sepilli cites twelve cases of atrophy of the cerebellum. These do not include the two recently described by Dr. Donald Fraser in the 'Glasgow Medical Journal' for March, which were probably published after Seppilli's paper was written. His observations certainly confirm the prevalent view that the cerebellum has something to do with the co-ordination of the different muscular adaptations necessary to maintain the equilibrium and harmony of the bodily movements. In the cases of Combette, Verdelli, Otto, and Lallement, there was no deficiency of co-ordinating power. In the latter case the atrophy was confined to one side of the cerebellum, and the median lobe remained unaffected, and according to Nothnagel it is this median lobe whose affection leads to the disturbance of co-ordinating power; but in Otto's case the cerebellum was very small, though apparently of healthy texture. Sepilli observes that in four cases of atrophy of the cerebellum (those of Combette, Fidler, Verdelli, and Otto) the intelligence was limited. In the two cases of Dr. Fraser the intelligence was not affected.

It is clear that there are still contradictions in our observations upon disease or deficiency of the cerebellum which cannot at present be harmonised. We have not yet reached a generalisation which will at once explain the function of the organ and enable us to understand the symptoms described as following upon diseased action in its texture.

Riva on Decolorisation of the Choroid in the Insane.—Dr. G. Riva (*Rivista Sperimentale di Freniatria*, Anno V. fascicolo iv.) claims to have discovered a true and special morbid process in the insane. This seems to consist in a diminution of the amount of pigment in the choroid coat, so that through the ophthalmoscope the papillæ appear paler than usual.

Dr. Riva found this condition present in thirty cases out of 117 lunatics studied by him. It seems especially common in pellagrous insanity. Lombroso, in his work on Pellagra, treated it as a phy-

siological process, the result of the depigmentation attendant on the coming on of old age. Riva regards it, at least in the degree observed by him, as peculiar to insanity. He sums up as follows:—The posterior chamber of the eye presents a characteristic appearance in insanity, especially in its intermittent and paroxysmal forms, and where it is dependent upon the pellagrous cachexy. This appearance is caused by diminution in the amount of pigment in the choroid coat and the epithelial pigment of the retina (*epitelio pigmentato retinico*), so that the retina becomes discoloured and turbid.

2. This is not a physiological phenomenon, since it is never observed in so prominent a degree in the sane.

3. The coincidence of these alterations in the fundus of the eye with morbid changes in the brain liable to produce acvtie or passive congestion in the vessels of the choroid, leads us to admit that hyperæmia, which in other parts of the body gives rise to hardening of the tissues, is the most frequent cause of the appearance described.

W. W. Ireland.